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General Practice Series

RECENT ADVANCES AND NEWER CONCEPTS IN THYROID DISORDERS

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In a review of thyroid disease published in this series[1] in 1957 some of the basic clinical concepts were discussed. It is the intention of this article to comment on some newer aspects. Many of these will appear to be of academic interest only, but they have important practical implications which will be considered.

PHYSIOLOGY

Ingested iodine is rapidly absorbed into the blood-stream, whence it is trapped by the thyroid gland or excreted into the urine (apart from small quantities which may be lost in the sweat or the faeces). In the thyroid gland the iodine is converted into thyroid hormone by a series of complex and incompletely understood reactions. The hormone is thought to leave the gland as free thyroxine which, when it re-enters the circulation, becomes attached to specific plasma proteins. Much interest has been taken recently in a substance called tri-iodothyronine which some people believe to be the compound responsible for the peripheral activity of thyroid hormone. It is thought to be derived from thyroxine, which possesses an extra iodine atom. While many claims have been made for the added therapeutic efficiency of tri-iodothyronine, there is as yet no real proof that its action is superior to that of thyroxine.

The release of hormone from the thyroid gland is controlled by the anterior pituitary secretion of TSH (thyroid stimulating hormone or thyrotropin). By the familiar feed-back mechanism the pituitary responds to low levels of circulating thyroid hormone by extra secretion of TSH, which stimulates the thyroid gland to extra activity; conversely, high circulating levels damp down pituitary action until it is needed again. In this way a balance is maintained between the utilization and production of thyroid hormone.

THE PATHOGENESIS OF GOITRE

Under certain circumstances the synthesis of thyroid hormone can be blocked or slowed down so that the blood levels fall. If the block persists, pituitary stimulation continues in a vain effort to restore these levels to normal. In such a case hyperplasia of the thyroid gland may result without corresponding increase in its output. This concept of hyperplasia with hypofunction is a most important one, the counterpart of which may be seen in the condition of congenital adrenal hyperplasia.

The *antithyroid drugs* (thiouracil compounds and methimazole) act in the manner considered above, i.e. by interfering with the normal synthesis of thyroid hormone. If their blocking action is prolonged and complete, hyperplasia of the thyroid gland results—hence the term 'goitrogen' applied to substances which impede the production of thyroid hormone. If the thyroid gland increases in size during the administration of antithyroid drugs, the dosage is probably excessive and should be reduced. Alternatively, thyroid sicca (or equivalent thyroxine or tri-iodothyronine) may be added with the object of restoring the blood hormonal levels to normal and abolishing the overactivity of the pituitary gland.

The same mechanism seems to prevail in the pathogenesis of spontaneous non-endemic *cretinism* or *hypothyroidism with goitre*. In many of these patients a similar block in thyroxine synthesis appears to exist—perhaps through deficiency of an essential enzyme. While the precise nature of the defect appears to differ from case to case, the end-result is the same in all, namely hyperplasia of the thyroid gland with low blood levels of thyroid hormone. Administration of thyroid sicca or thyroxine restores the latter to normal, combats the hypothyroidism and damps down the pituitary activity. Naturally in these goitrous hypothyroid subjects therapy must be continued for life.

While iodine deficiency is fundamentally the cause of *endemic goitre*, interest has been aroused in the role of naturally-occurring goitrogens in some areas. An early report described an 'outbreak' of goitre amongst war-time internees. Turnip was identified as the agent responsible. A mild antithyroid action has long been attributed to this vegetable; when it became a staple food, this action was magnified to the point at which it interfered with thyroxine synthesis and goitre resulted. The seeds of cabbage, which is also a member of the brassica family, have been incriminated and from Tasmania has come a report of goitre occurring amongst schoolchildren who had ingested milk from cattle fed on thousand-headed kale.

An interesting sidelight on the pathogenesis of endemic goitre has been the realization that goitre does not occur in all inhabitants of an endemic area, but tends to concentrate in certain families. This suggests that a genetic factor might exist with regard to adaptation to iodine deficiency.

HASHIMOTO'S THYROIDITIS

A similar but more involved mechanism seems to account for the development of this condition. Recent work has given rise to an entirely new concept of auto-immunization, which may prove important in the pathogenesis of many

other unrelated disorders. Normally after birth the body forms antibodies only to foreign substances introduced into the blood-stream from without. Certain body proteins do not normally enter the blood-stream, e.g. spermatozoa, lens protein and thyroglobulin (the protein to which thyroxine and other iodinated compounds are attached in the thyroid gland). If these substances do reach the circulation, they may stimulate the production of antibodies in the manner of foreign proteins. This process of antibody reaction to the host's own proteins is known as auto-immunization.

In Hashimoto's disease this mechanism has been postulated. Under certain circumstances thyroglobulin escapes into the blood-stream and antibodies develop. These not only destroy the circulating thyroglobulin, but attack that which is still contained within the thyroid gland. This leads to gradual loss of thyroid function, with reduced formation of thyroid hormone. Again the pituitary exerts its stimulatory effect; hyperplasia with hypofunction results. Clinically the hypothyroidism may not be marked until a late stage of the disease, but it is important to consider this condition in cases of non-toxic goitre. Since operation will aggravate the hypothyroidism, it is only indicated where there is evidence of considerable compression of surrounding structures. The correct treatment is administration of thyroid hormone, which restores the blood levels and allows the thyroid to involute. Therapy must be permanent.

HYPOTHYROIDISM IN CHILDREN

Far too many errors are still made in the diagnosis of this condition. Thyroid hormone is still being used widely in the treatment of obesity in children whose only fault is gluttony. At times these preparations are used as non-specific slimming agents—a course that cannot be justified on scientific grounds; otherwise they are administered in the mistaken belief that obesity (perhaps with somnolence) is the result of thyroid underactivity. A plea must be made for accurate diagnosis in these cases. Thyroid hormone is a potentially dangerous drug and its indiscriminate use must be deplored.

Of greater consequence are the numbers of truly hypothyroid children whose disease is not recognized, where the correct use of thyroid hormone might be crucial to their proper development. Even with the full range of modern techniques the correct diagnosis may not be easy, but every effort should be made to substantiate it at the earliest possible age.

While the thyroid gland is generally atrophic and therefore impalpable, an enlarged gland does not exclude the diagnosis and may, in fact, be a point in its favour. In early post-natal life clues may lie in such complaints as lethargy, constipation, feeding problems, and respiratory difficulty. Laboratory tests may be misleading although, as a rule, the serum cholesterol is raised and the serum alkaline phosphatase lowered. Bone age is probably invariably retarded—a finding more specific for hypothyroidism in the younger patients. Stable protein-bound iodine estimations and radio-iodine tests may prove valuable, but many observers feel that the latter should not be employed in small children because of risks of carcinogenesis. This objection may be overcome by the use of small doses of shortlived isotopes, e.g. ^{132}I in preference to ^{131}I.

The therapy of childhood hypothyroidism has also come under recent review, there being many who believe that under-treatment is prevalent and who urge the use of thyroid hormone in doses which approach the patient's limit of tolerance. Certainly there can be no rule-of-thumb guide to dosage and adequacy must be measured by definite indices. Serum cholesterol and protein-bound iodine levels are helpful guides. Bone age should be assayed regularly and should approach chronological age. This cannot take place until the height of the serum alkaline phosphatase overshoots the adult range and enters that of the normal growing child. About a month of therapy is required before this effect is noted. Average doses for infants would range from 2-3 grains of thyroid sicca (approximately 0·2-0·3 mg. of thyroxine or 50-75 micrograms of tri-iodothyronine); larger children may need higher doses. Perhaps with these larger doses we may see fewer of those therapeutic failures which we have previously attributed to 'irreversible damage due to long-continued thyroid deficiency'.

TESTS OF THYROID FUNCTION

Determination of the *serum-cholesterol* level remains a useful test for hypothyroidism, but not for hyperthyroidism. It is, however, probably the least reliable of the many tests of thyroid function.

The *basal metabolic rate* requires skill in performance and in interpretation. In proper hands it is a valuable guide—greater importance being attached to low than to high readings.

Estimation of the level of *protein-bound iodine* in the serum can only be done at certain specialized centres, but serum can be sent to these when necessary. This is probably the most valuable single parameter of thyroid function, since it is in effect a direct measure of the level of circulating thyroid hormone. Unfortunately many artefacts may contribute to false readings, amongst these being recent use of mercurial diuretics or of organic iodine-containing radiographic contrast media or inorganic iodides. The normal range for adults is 4·0 to 8·0 μg per 100 ml, and is somewhat higher during pregnancy and in the first few weeks of life.

Tests employing *radio-active iodine* depend upon the introduction into the body of minute amounts of iodine bearing a 'label' of radio-activity which allows reasonably accurate assay of infinitesimal amounts. The patient drinks a colourless, odourless, tasteless liquid which contains the iodine and reports back at specified times for 'counting' over the thyroid region. This procedure takes only a few minutes. In some centres urine studies are done (since renal excretion varies inversely with thyroid activity) and blood analysis. In this way measurements can be made of the rates at which the thyroid concentrates and discharges the iodine. The major drawbacks to this procedure are the necessity of trained staff and specialized equipment and, again, invalidation of the results by previous drugs and therapy. Any iodine-containing compound—even iodized salt and cough preparations with potassium iodide, any antithyroid agent or thyroid substance, will interfere with the test, which may also be of least value where it is most needed, e.g. after previous thyroidectomy, in mild cases of hyperthyroidism and in some cases of nodular goitre.

Used intelligently these tests can be of immense help in the diagnosis of thyroid dysfunction; knowledge of their deficiencies can only increase their value.

THERAPY IN THYROID DISEASE

Other than the introduction of radio-iodine—which can no longer be considered new—there have been no major advances in this sphere. For *hyperthyroidism* there remain 3 methods of treatment:

1. *Prolonged medical treatment*, i.e. the use of antithyroid drugs for periods of at least a year. Success with this regime is more likely with careful selection of patients, the most suitable being young females with small goitres of a diffuse type and mild or moderate hyperthyroidism. If medical treatment is confined to this group there is a cure rate of about 70%, but high relapse rates after cessation of therapy hardly justify its use in other cases.

2. *Surgery*. Despite the illogicality of this sort of operation sub-total thyroidectomy still offers most chance of permanent cure and is probably the treatment of choice for most types of hyperthyroidism.

3. *Radio-iodine*. The results of more than 16 years' experience have helped to clarify its indications. Suitable patients include those with recurrent thyrotoxicosis following operation and those in whom medical treatment has failed and where there is a contra-indication to operation, but it may be preferred in many other hyperthyroid patients who lack these indications and who are over the age of 45 years. Pregnancy is a complete contra-indication to its use, and younger patients should preferably be treated by other means in view of the theoretical risks of carcinoma. The treatment is exceedingly simple and cure can almost be guaranteed, although probably not more than about 60% of patients respond to the first dose. For this reason and the high incidence of post-therapy hypothyroidism (10-15%) regular follow-up visits are essential. It should be noted that while hypothyroidism generally occurs within 6 months, it may appear slowly and insiduously 5 years or more after treatment. This must be borne in mind by practitioners attending patients who have received this form of therapy.

The *eye-signs of thyrotoxicosis* generally consist of lid-lag and lid-retraction with minimal exophthalmos. Occasional hyperthyroid patients show more extensive eye-signs, including more severe degrees of exophthalmos, diplopia, ophthalmoplegia and oedema. In others the eyes deteriorate during anti-thyroid drug administration or following surgical or ^{131}I therapy. Great care must be taken to ensure that these patients do not become hypothyroid as a result of excessive treatment, since this state seems to aggravate existing eye signs. In all such patients small amounts of thyroid hormone or thyroxine (2-3 grains of the former or 0·1-0·3 mg. of the latter per day) should be given with the antithyroid drug. Many people contend that similar thyroid therapy should follow all surgical or radio-iodine treatment of hyperthyroidism. This approach seems perfectly reasonable, since it may prevent the development of severe eye complications.

In the treatment of adult *hypothyroidism* 3 grains of thyroid sicca (or equivalent) generally suffices. When this dosage appears ineffective the validity of the diagnosis should be reconsidered. A point worth noting here is that occasional batches of thyroid sicca have proved to be inactive. We have certainly experienced this with locally produced hormone. Replacement with thyroxine in equivalent dosage has resulted in therapeutic response. The extra cost of thyroxine is so slight that we use it as a routine in preference to thyroid sicca.

No ready solution exists for the problem of *non-toxic nodular goitre*. Single nodules are usually removed because of a greater risk of carcinoma, although selection may be made on the grounds of radio-iodine tests. Those nodules which concentrate iodine readily are less likely to be carcinomatous, while those that are 'cold' should certainly be removed. 'Hot' nodules may be treated with thyroid hormone with a fair chance of shrinkage. Goitres which are very large may be removed for cosmetic reasons of for pressure effects on neighbouring structures.

Small non-toxic multinodular or diffuse goitres are probably best left alone or at most treated with thyroid hormone. Reassurance is often necessary, since many patients are afraid that the goitre will either grow inwards and choke them, or will turn into cancer. Final decision often depends on the skill and availability of the surgeon and on the possibilities of follow-up from the social and economic points of view.

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ELEKTROKARDIOGRAFIESE STUDIES III

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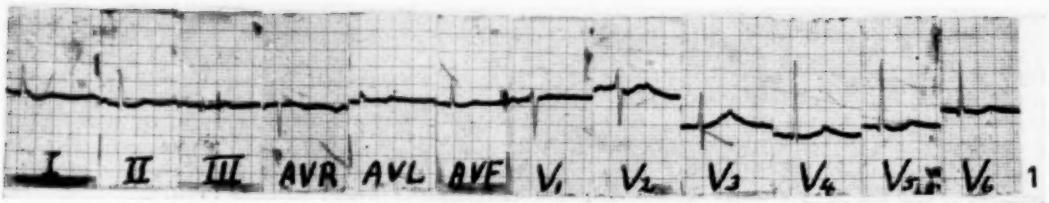
Geval 3. Vertraagde Verskyning van Elektrokardiografiese Beeld van 'n Hartinfarkt van die Agterwand

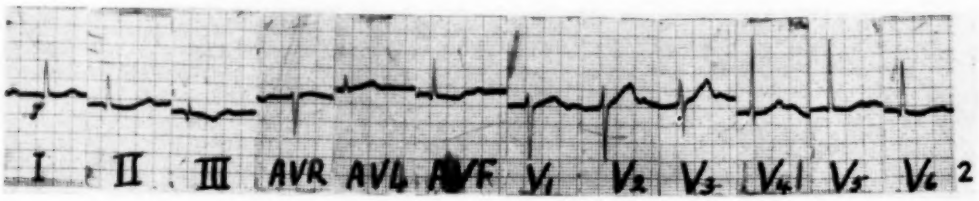
Die pasiënt is 'n 41-jarige Blanke man met 'n geskiedenis van substernale pyntoevalle met inspanning wat 6 weke voor toelating begin het. Die toestand het weer verbeter, en selfs verdwyn, maar ongeveer 'n week voor sy opname het soortgelyke pyntoevalle egter weer voorgekom. Die aanvalle het toegeneem en hy het die dag voor toelating 'n langdurige, hewige pyntoeval gekry wat substernaal geleë was. Dit het met rus aangekom en ongeveer 'n uur lank geduur. Gedurende dieselfde nag het hy weereens pyn in die borskas gehad wat 'n halfuur lank geduur het; toe het dit bedaar, en hy het geslaap. Die oggend wat hierop gevolg het, en die dag van toelating, het hy aan die ontbyttafel 'n toeval van floute gekry en gevolglik sy geneesheer gaan spreek. Daar was by sistematiese navraag geen ander simptome nie.

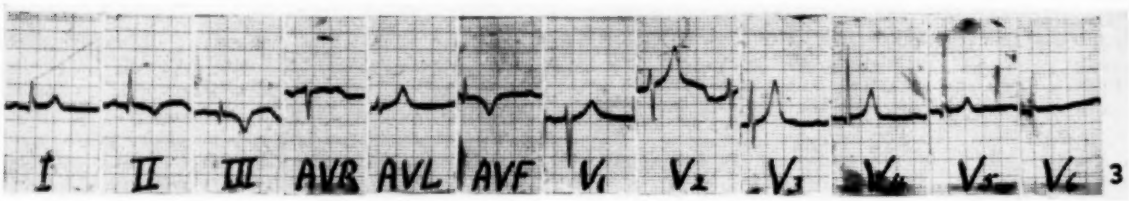
By fisiese ondersoek was daar nie enige afwykings van betekenis nie, behalwe dat die bloeddruk slegs 100/70 was; ook het hy 'n bradikardie van 45 per minuut gehad. Andersins het die pasiënt goed daar uitgesien. Urine-ondersoek het geen afwykings gelewer nie. Die hemoglobien was 14·2 g.% (96%), rooibloedseltelling 4,500,000 per c.mm., witbloedseltelling 5,850 per c.mm. en bloedbesinking 7 mm. in die eerste uur. Die differensiële telling was normaal. Daar was geen verhoogde temperatuur nie.

ELEKTROKARDIOGRAMME

Afb. 1: Elektrokardiogram geneem op die oggend van toelating tot die hospitaal: Sinusritme 65 per minuut. Daar is geen geleidingstoornis nie. QRS-komplekse kom normaal voor. Twyfelagtige verbreding van Q, 0·03 sek. in standaardafleidings 3 en aVF. Daar is matige afsakking van die ST-segmente in aVF







en V4 tot V6. Die T-uitwykings is van lae spanningshoogte, maar binne normale perke.

Gevolgtrekkings

Die elektrokardiogram dui matige ischemie oor die linkerventrikel aan, maar is origens binne normale perke. 'n Kardiogram, 24 uur later geneem, was onveranderd.

Afb. 2: Elektrokardiogram 48 uur na opname geneem. Spoed en ritme onveranderd. Daar is nou 'n meer prominente Q, 0·04 sek. in standaardafleidings II en III, asook aVF met 'n negatiewe T-uitwyking in standaard III en aVF. T-uitwykings in prekordiale afleidings V2 en V3 is verhoog in vergelyking met die uitwykings in die vorige afbeelding. Verder is daar nog die voorkoms van ischemie in V4 tot V6.

Gevolgtrekkings

Daar is nou duidelik elektrokardiografiese bewys van 'n kardiale infarksie van die agterwand.

Afb. 3: 14 dae na toelating tot die hospitaal: Hier is die voorkoms van 'n hartinfarkt van die agterwand elektrokardiografies nou nog meer prominent, met die kenmerkende Q en T veranderinge in II, III aVF en verhoogde T-uitwykings in die prekordiale afleidings V2, V3 en V4.

BESPREKING

Die pyngeskiedenis van hierdie pasiënt het kardiale ischemie, en moontlik infarksie, aangedui. Die betreklike lae bloeddruk het ook 'n moontlike infarksie aangedui. Daar was geen ondersteunende bevindings, soos 'n versnelde bloedbesinking of 'n verhoogde witbloedseltelling nie. Die eerste elektrokardiografiese opname het ook net 'n geringe ischemiese verandering aangetoon. Na 48 uur was die kardiografiese beeld van 'n hartinfarkt van die agterwand duidelik waar te neem, en het met verloop van tyd nog meer prominent geword.

Dit is dus duidelik dat die pasiënt se siektegeskiedenis, saam met 'n bloeddruk aan die onder-grense van normaal, genoegsame redes was om opvolg-elektrokardiogramme te neem. Ten spyte van die afwesigheid van verdere episodes van pyn, het die beeld van agterwand-infarksie tog na 48 uur te voorskyn gekom.

Opvolg-ondersoeke is nie noodsaaklik as daar reeds elektrokardiografiese bewys gelewer is van 'n infarksie nie. Wanneer infarksie egter nog nie gedemonstreer is nie, moet daar opvolg-grafieke gemaak word totdat 'n diagnose bereik is, of 'n ander diagnose in sy plek gestel kan word. 'n Tydperk van 3 tot 4 dae voor die verskyning van die diagnostiese beeld is nie seldsaam nie. Levine[1] vermeld selfs 'n geval wat 3 weke na die kliniese episode eers elektrokardiografiese bewyse van infarksie opgelewer het.

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EDITORIAL : VAN DIE REDAKSIE

THE DIAGNOSIS OF ANAEMIA

We are sure that many of our readers will remember the days when the red cell count was regarded as one of the important investigations in the diagnosis of anaemia. The red cell count together with the haemoglobin level enabled us to calculate the 'colour index' and, taken with the haemotocrit level, the mean cell volume could be determined. If the colour index (or mean cell volume) was low, deficiency of iron could reasonably be postulated, while if these indices were elevated the anaemia would probably respond to injections of liver. These rough guides worked reasonably well in practice but they undoubtedly engendered a false sense of accuracy. If a substantial error occurred in any of these basic tests the 'absolute index' would probably be incorrect.

It has been shown that considerable errors occur in the red cell count.[1, 2] Biggs and MacMillan,[2] for example, calculated that the standard error of the red cell count lay between 8 and 10%. Errors arose from personal bias in counting and in the selection of counting areas, as well as from the random distribution of cells in the counting chamber. Many of these errors arise because it is seldom possible to count more than 1,000 cells in a single count. The recent introduction of electronic red cell counters has gone far in eliminating many of these errors. One model, for example,[3] can count about 10,000 cells in 2 minutes with a high degree of accuracy. If a reliable estimation of haemoglobin can be made on the same sample of blood there is virtue in the calculation of the colour index. The normal colour index thus estimated has been found to be 0·99[4]—a very surprising and apparently fortuitous approximation of the so-called normal figure of 1 which was calculated by less accurate methods many years ago.

The estimation of haemoglobin has presented many difficulties. Because of its great importance the Medical Research Council (London) appointed a Committee to investigate the matter. They found that reliable results could be obtained by the use of an oxyhaemoglobin method using a neutral grey wedge photometer.[5, 6] Reliable results can also be obtained by the use of cyanmethaemoglobin solutions read in a photo-electric colorimeter.[7] The introduction of a service by which blood of known haemoglobin concentration is made available to any laboratory requesting it, has done much to simplify standardization. Colour standards for cyanmethaemoglobin are also commercially available. However, all methods for the estimation of haemoglobin require great care, and a simple, inexpensive and completely reliable instrument suitable for use by the average practitioner in his office still eludes us. None of the instruments at present available can be relied on to give really accurate estimations under these circumstances.

The haematocrit estimation has been the most reliable of the three basic methods and has been widely used. The technique described by Wintrobe[8] in 1929 using venous blood has gained almost universal acceptance and is regarded as one of the simplest and most reliable of methods. Biggs and MacMillan,[9] for example, found the coefficient of variation to be only 1%. This valuable tool, however, has had the drawback that it usually requires venous blood which is not always readily available especially in infants and small children. Methods using capillary blood have long been available but have not gained wide acceptance. The recent introduction of a centrifuge specially designed for this purpose is likely to reawaken interest in this technique. Disposable capillary tubes containing anticoagulant are used and these only require very small samples of capillary blood. The technique is simple, requires only five minutes of centrifugation, and gives accurate results that are virtually interchangeable with Wintrobe readings.[10] It is even possible to adapt some existing centrifuges for this purpose.

A reliable haemoglobin reading combined with an accurate measure of the haematocrit enables an estimate of mean corpuscular haemoglobin concentration to be made. A low MCHC is the key to iron deficiency which is still a very common cause of anaemia (if not the commonest cause). That this index can now be calculated easily, rapidly and accurately from a drop of blood obtained by a prick of the finger represents a distinct advance in haematological diagnosis.

Even the technique of pricking a finger has not been neglected in this era of technical advance. It is to be hoped that the needle immersed in spirit and used on all and sundry has been superseded for all time by the availability of disposable needles. Sterile, sharp and individually wrapped, they are meant to be used only once. No longer need one fear the transmission of viral hepatitis, and the cost of a penny or two is more than compensated for by the elimination of this hazard.

It is not anticipated that electronic red cell counters will be used otherwise than as a research tool for some time to come; in any case the estimation of the red cell count is not of greater help in the diagnosis of the cause of anaemia than these other more readily available techniques. Cases of anaemia often require more detailed haematological diagnosis. But the demonstration that the patient is anaemic and that the anaemia is probably due to deficiency of iron as shown by a low MCHC is a good first step in the diagnosis of a common disease. Provided one is reasonably certain that the patient is not bleeding, there is no harm and often much virtue in a single course of oral iron therapy.

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DORS EN WATER

Gesonde en siek persone ondervind meermale per dag die sensasie van dors—'n toestand wat vergelyk kan word met honger, en wat slegs deur die inname van vloeistof geles kan word. By die siek persoon is daar egter velerlei faktore teenwoordig wat hierdie sensasie, nie net meer dikwels laat voorkom nie, maar soms ook ernstige afmetings laat aanneem.

Daar is byvoorbeeld die pasiënt wat deur sy mond asemhaal en sodoende die mond self uitdroog. Daar is ook die koorsige pasiënt, en die pasiënt met verlies van vloeistof en elektroliete, byvoorbeeld die diarree-lyer. Daar is gevind dat dors soms die aanvang van diarree voorafgaan as gevolg van die verlies van isotoniese vloeistof deur die lumen van die derm met vermindering van die ekstrasellulêre volume.[1] Nadat groot volumes vloeistof uit die pleurale of abdominale holte verwyder is, is dors 'n baie algemene simptoom. Om op te som sou ons kon sê dat 'n styging in die osmotiese druk van liggaamsvloeistowwe *mag* lei tot dors selfs sonder volumeveranderings, maar dat veranderings in die volume van die verskillende vloeistof-kompartemente *altyd* lei tot dors.[2] Dikwels word die pasiënt ook aangemoedig om baie water in te neem, byvoorbeeld na urologiese operasies.

Juis hierdie faktore maak dit noodsaaklik dat ons noukeurig moet let op die drinkgewoontes van die pasiënt en die aard van sy vog-inname. Dit is dus ontstellend om te verneem dat oor die waterkraffies in vier-en-twintig Bostonse hospitale gesê is: ‚growwe onhigiëniese toestande is gevind in twee derdes van die kraffies. Dooie, gedeeltelik ontbinde insekte is dikwels gevind. Die wande van sommige kraffies was slymerig; gelatineuse eilande van alge en fungi het in ander rondgedryf.'[3]

Die suig van water deur 'n strooitjie word blameer vir die terugvloei van speeksel in die water, terwyl die omkeer van 'n glas oor 'n waterbottel dieselfde effek mag hê.[3] Die vorm van baie waterbottels met 'n nou nek bemoeilik deeglike reiniging daarvan.

Hoe toestande elders in die wêreld gesteld is, is nie bekend nie, maar dit sou dui op 'n aspek van hospitaalhigiëne wat ons aandag verg.

Gardner,[4] in 'n studie oor die aspirasie van kos en vomitus, stel voor dat die standaard hospitaal-voedingsbeker, ontwerp om die voeding van bedlêende pasiënte te vergemaklik, verwerp moet word. Die ontwerp en styl van sy spuit is van so 'n aard dat dit moontlik is om vloeistof in die trachea af te gooi, en die bedekte rand lei daartoe dat die pasiënt onverhoeds gevang word deur vloeistof in sy mond. Die pasiënt moet òf sit en uit 'n gewone glas drink, òf deur 'n strooitjie die water opsuig. So nie, moet 'n maagbuis gepasseer word en alle vloeistof daardeur toegedien word.

Hierdie oorwegings laat mens aan die woorde van die drenkeling op see dink: ‚Water, water orals rond en nie 'n (suiwer) druppel om te drink nie.' 'n Oplossing, hoewel dit maklik lyk, kan ons nie gee nie. Die minste wat ons egter kan doen is om seker te maak dat die dorstige pasiënt skoon, vars water kry om te drink.

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POLIOMYELITIS

Poliomyelitis continues to attract world-wide productive research. It is fitting that this knowledge was shared at an international poliomyelitis conference and that the papers and discussions presented at the Conference in Geneva (July 1957) are published in book form.

In this symposium the magnitude of the problem of poliomyelitis is reported by delegates of many countries. Hence an effective vaccine capable of preventing the increasing epidemics and crippling paralysis has become a matter of priority. Every basic step such as tissue culture, viral multiplication, etc. is given detailed description to aid the production of a vaccine of this nature. Important research results compare the immunizing value of the orally administered, attenuated live-virus vaccines of Koprowski and Sabin, with the killed-virus Salk vaccine. The Salk vaccine has now been extensively used and attenuated live-virus vaccines have since undergone successful field trials in the Belgian Congo.

The Coxsackie and Echo groups of enterogenous viruses receive detailed attention not only because they have to be distinguished diagnostically from poliovirus, but because they are increasingly associated with diverse clinical entities, some of which simulate poliomyelitis.

Clinically, the new advances in the treatment of paralysis and respiratory involvement are well described. These methods allow the survival of many patients who, previously, would have died. The rehabilitation of these severely incapacitated victims of poliomyelitis by orthopaedic and other means receives urgent consideration. Survival without rehabilitation would appear wasteful and cruel.

Poliomyelitis is a magnificent book in which every aspect of polio is treated by an acknowledged world authority. No library or interested postgraduate should be without it.

1. Fourth International Poliomyelitis Conference, 1957 (1959): *Poliomyelitis*, Philadelphia and Montreal: J. B. Lippincott Co.

JOHN DONNE ON HIS OWN DESTRUCTION*

Fevers upon wilful distempers of drinke, and surfets, Consumptions upon intemperances, and licentiousnes, Madnes upon misplacing, or overbending our natural faculties, proceed from our selves, and so, as that our selves are in the plot, and wee are not onely passive, but active too, to our owne destruction; But what have I done, either to breed, or to breath these vapors? They tell me it is my Melancholy; Did I infuse, did I drinke in Melancholy into my selfe? It is my thoughtfulnesse; was I not made to thinke? It is my study; doth not my Calling call for that? I have don nothing, wilfully, perversly toward it, yet must suffer in it, die by it; There are too many Examples of men, that have bin their own executioners, and that have made hard shift to bee so; some have alwayes had poyson about them, in a hollow ring upon their finger, and some in their Pen that they used to write with: some have beat out their braines at the wal of their prison, and some have eate the fire out of their chimneys: and one is said to have come neerer our case than so, to have strangled himself, though his hands were bound, by crushing his throat between his knees; But I doe nothing upon my selfe, and yet am mine owne Executioner.

* *Complete Poetry and Selected Prose.* By John Donne.

MECAMYLAMINE AND RESERPINE IN THE MANAGEMENT OF SEVERE AND MALIGNANT HYPERTENSION IN UGANDA

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It used to be the impression of a number of clinicians that hypertensive disease was uncommon in the indigenous peoples of East Africa. This view was certainly refuted by Williams (1944) and more recently by Leather (1958), who, working in Uganda, was able to select 49 new cases of hypertension for study in a period of less than 14 months. Faced with an increasing recognition of the frequency of severe forms of hypertensive illness and at the same time appreciating the established value of ganglion-blocking compounds, alone and in combination with rauwolfia alkaloids, in the treatment of malignant and non-malignant hypertension (McMichael and Murphy, 1955; Doyle and Smirk, 1955; McQueen and Smirk, 1956), we decided, early in 1958, to embark on a regime of management of hypertension on proper clinical lines at Mulago Hospital, an African hospital in Uganda.

Experience gained elsewhere (Milne *et al.*, 1957), and generally satisfactory reports of its use at other centres (Doyle *et al.*, 1956; Smirk and McQueen, 1957; Kitchin *et al.*, 1957), resulted in the choice of mecamylamine for use at this clinic. The reports of Freis (1955), Ford *et al.* (1956) and Milne *et al.* (1957) have dealt in detail with the pharmacology of mecamylamine.

The present study describes the result of our experiences with the use of mecamylamine in combination with reserpine in the management of 29 cases of hypertensive disease treated for periods ranging from 3 months to 9 months.

Aetiology and Incidence of Hypertensive Disease

Mulago Hospital is the largest and most centrally placed hospital in Uganda. Its intake reflects a wide variety of conditions. In a single year's survey (Shaper and Shaper, 1958), hypertension constituted the largest single group, totalling 22% of all cardiovascular admissions in the medical wards of the hospital. That the greater part of cases of hypertension is due to chronic renal disease rather than essential hypertension was first noted by Williams (1944). Of the 49 cases of hypertension studied by Leather (1958), 29 were considered to be renal in origin. There were only 14 cases of essential hypertension, and in the remaining 6 cases aetiology was uncertain. It is therefore not unexpected that this treatment series includes a majority of cases with renal rather than essential hypertension.

Selection of Cases

In the selection of cases, besides generally agreed criteria, viz. high blood pressure associated with objective evidence of hypertensive retinopathy, electrocardiographic changes of left ventricular hypertrophy, or heart failure, several factors of local importance had to be taken into consideration. Some patients, who because of their simple backgrounds were unlikely to appreciate the importance of follow-up after initial improvement on treatment, proved unsuitable cases for long-term management. Others, who were migrant or returned to their homes in remote areas, also failed to attend clinic. Several patients, however, attended regularly despite distances of 40 miles or more by rural transport. Literacy and sophistication did not seem important in the selection of cases, and in practice intelligent patients with a sense of cooperation living in and around the immediate districts of Kampala were the most suitable cases for follow-up.

No patient with mild or asymptomatic hypertension was advised treatment. Nine patients had had treatment with other ganglion-blocking drugs previously. In the remainder treatment was instituted with mecamylamine. All the patients were Africans; they belonged to a variety of tribal groups found in Uganda.

Results of treatment are only considered in 24 patients. Of these, 6 were treated for 6 or more months, and 18 for 3 or more months. In 4, treatment was abandoned because of disabling side-effects of treatment. The 24 included 9 males and 15 female subjects, of ages ranging from 20 to 54 years. Five other cases were excluded from the study because of default in attendance or inadequate follow-up due to factors mentioned above.

Assessment of Cases

All the patients were admitted into hospital for initial assessment. Special investigations were directed towards detecting cases with chronic renal disease. Essential hypertension was diagnosed by exclusion.

The following factors were investigated in all cases: Urine (including culture and 24-hour collections for total proteinuria), blood urea, electrocardiograms, chest radiography and fluoroscopy, and fundal examination. In the absence of gross proteinuria, urine concentration test was performed, and in those cases where blood urea was not grossly raised, intravenous pyelography. Where a past or micturition history suggested the possibility, urethral catheterization was done to exclude urethral stricture, a not infrequent causative factor in secondary hypertension in Uganda. Renal biopsy was carried out in 11 cases.

Fundal changes were graded 1 to 4 (Keith *et al.*, 1939). The term malignant hypertension was applied to patients with grade-4 fundal changes. Electrocardiographic evidence of left ventricular hypertrophy was graded from 0 (absent) to 4, the four positive grades representing the common English adjectives of degree, slight, moderate, considerable and gross.

A diagnosis of chronic pyelonephritis was made on consideration of the following grounds: A history of acute pyelonephritis, untreated gonorrhoea or urethral stricture; pyuria and bacteriuria; loss of urinary concentrating power, with blood-urea levels within normal limits (Raaschou, 1943). Chronic glomerulonephritis was diagnosed on the presence of persistent proteinuria, with the passage of large numbers of casts. No reliable history of acute glomerulonephritis was forthcoming. Even on applying these simple

* In receipt of research grants from Makerere College and Uganda Medical Department.

clinical criteria, chronic pyelonephritis and chronic glomerulonephritis might readily have been overlooked.

In the 11 cases in whom renal biopsy was carried out an accurate diagnosis was possible.

CLINICAL FINDINGS

Certain important features emerge in the series. There were 13 cases which presented with congestive heart failure and 3 with left ventricular failure. All but one case of congestive heart failure showed grade-3 or grade-4 electrocardiographic changes of left ventricular hypertrophy. Five of these were cases of malignant hypertension, of whom 3 showed evidence of renal failure in that blood-urea levels at the time of diagnosis of the malignant phase persistently exceeded 100 mg. per 100 ml.

There were only 5 patients who presented with headache as the only clinically important symptom, and one of these had malignant hypertension. No cases of major cerebrovascular accidents were selected for treatment. One patient with malignant hypertension presented with loss of vision.

Three patients were diagnosed during pregnancy, 2 with toxaemia and one with recurrent left ventricular failure in the last trimester.

Aetiologically, 11 cases were considered to be due to chronic pyelonephritis, 2 to chronic glomerulonephritis, and 3 to pregnancy toxaemia. There were 8 cases of essential hypertension and one doubtful essential. One patient (case 21), also had mitral and tricuspid incompetence, which was considered to be due to endomyocardial fibrosis, a common cause of valvular disease described from Uganda (Ball *et al.*, 1954; Davies and Ball, 1955).

For reasons explained, the series represents a highly selected group of cases of severe hypertensive disease with undoubted indications for treatment; the proportions of aetiological types are therefore not necessarily representative of the general incidence in Uganda.

The important clinical facts relating to each case are given in Table I.

Measurement of blood pressure. In the ward, blood-pressure readings were taken for several days to obtain basal levels before treatment. The exceptions were cases of malignant hypertension in whom treatment was started on an emergency basis. Once treatment was commenced, blood pressure was recorded with the patient first supine and then erect.

After discharge from hospital, patients were seen first at a week's interval and thereafter fortnightly or monthly as indicated by requirements of treatment and the patient's convenience.

ADMINISTRATION OF MECAMYLAMINE

As the duration of action of mecamylamine is long—more than 12 hours—it was decided to treat patients with two doses a day.

The initial dose was usually 2·5 mg. and it was increased by 2·5 mg. every other day until the blood pressure was adequately lowered. If the blood pressure remained high, the dose was increased by 5-10 mg. every other day. Dosage increments were more cautious in patients with chronic uraemia, as it is known that excretion of mecamylamine is delayed in patients with chronic renal failure (Milne *et al.*, 1957).

Initial stabilization was done in hospital. The dose was further adjusted at subsequent visits to the clinic.

Postural hypotension after administration of mecamylamine. The occurrence of postural hypotension after the administration of ganglion-blocking drugs has been referred to on many occasions. Whilst the extent of postural hypotension varied from patient to patient, our experience was similar to that of Kitchin *et al.* (1957) in that the benefit obtained by posture was often slight. Nevertheless, patients were advised to avoid the horizontal position as much as possible during the day and to raise the head of the bed at night.

Sensitivity and tolerance. The dose required to achieve a satisfactory fall of blood pressure varied widely from patient to patient. Many patients developed a material degree of tolerance in the early stages, necessitating periodic adjustments in dosage. The development of tolerance emphasized the need for regular supervision of treatment. The final stabilizing dose ranged from 5 mg. to 80 mg. (average 30·1 mg.) per day. Some patients were highly resistant and, in a few, resistance increased as dose was increased. Doses were increased gradually to avoid undue side-effects.

Use of reserpine in conjunction with mecamylamine. Advantage was taken of the beneficial effect of a combination of mecamylamine with reserpine. The dose of reserpine prescribed was 0·25 mg. twice daily.

RESULTS OF TREATMENT

Because of the somewhat exacting nature of the regime, therapy was limited to selected hypertensives whose illness was complicated by burdensome headache and dizziness, heart failure, or malignant hypertension. In the remaining majority, it was felt that prognosis had become sufficiently poor to strongly justify a regime that demanded intelligent cooperation by the patient and set in train side-effects creating at times difficult management problems. In general, a fall in blood pressure could not be regarded as the important criterion of successful treatment unless it was followed by the relief of complications. As with other hypotensive agents, however, satisfactory control of blood pressure often resulted in the alleviation of symptoms, regression of hypertensive retinopathy or the electrocardiographic pattern of left ventricular hypertrophy, and the relief of cardiac failure, in some cases permitting the elimination of maintenance digitalis. One patient (case 19) with toxaemia of pregnancy was successfully taken to term on treatment. Because of the short time for which most patients in this series were treated, greater reference will be made to the control of blood pressure and relief of disablement from heart failure and the malignant phase of hypertension. That moderate uraemia does not rule out successful management and in fact may regress on treatment, is indicated by the following history:

Case 2, B.K., a male tailor, Ganda, aged 26, was first seen in September 1958. There was a history of venereal infection—? nature in 1950. In 1956 he developed headaches. In January 1958 headaches had increased in severity and were soon followed by blurring of vision and vomiting. Recurrent vomiting and hiccoughs had been present for 2 days before admission.

On examination. Grade 4 retinopathy. Blood pressure 250/170 mm. Hg. Atrial gallop, left ventricular hypertrophy, raised jugular venous pressure, peripheral oedema. Blood urea 208 mg. per 100 ml. Urine: Albumen +++, many red cells and pus cells in deposit.

TABLE I. CLINICAL DATA AND RESULTS OF TREATMENT OF 24 PATIENTS TREATED WITH MECAMYLAMINE AND RESERPINE

Case No.	Sex	Age	Aetiology of hypertension	Major symptomatology	Fundi	ECG	Average blood pressure (mm. Hg)		Daily dose mecamylamine (mg.)	Total follow-up (mths.)	General result	Remarks
							Before treatment	After stabilization*				
1	F	47	Essential	Headache, dizziness	1	N	210/110	130/90	15	5	Good	Headache ceased
2	M	26	Chronic pyelonephritis	Congestive heart failure, renal failure	4	LVH (4)	250/170	130/110	10	4	Good	Regression of malignant phase and renal failure; no recurrence of heart failure
3	F	28	Chronic pyelonephritis	Headache	2	LVH (1)	190/130	110/90	7·5	6	Good	Headache ceased
4	M	49	Essential	Congestive heart failure	1	LVH (3)	250/150	140/90	50	5	Poor	Mecamylamine tremor. Treatment abandoned
5	F	25	? Essential	Left ventricular failure	2	LVH (2)	220/130	150/100	40	4	Good	No further LVF
6	F	38	Chronic glomerulonephritis	Congestive heart failure	1	NT	240/160	148/80	45	4	Good	Headache ceased; heart failure regressed
7	F	31	Pregnancy toxaemia	Headache, dizziness	N	LVH (1)	250/150	150/130	25	5	Poor	Tolerance; marked curare-like effects. Treatment abandoned
8	M	54	Essential	Left ventricular failure	1	LVH (4)	250/130	140/80	20	4	Good	No further LVF
9	F	41	Essential	Headache	4	N	230/140	150/100	15	8	Good	Regression of malignant phase. Tolerance; curare-like effects
10	F	50	Essential	Congestive heart failure	1	LVH (4)	210/140	130/90	60	4	Good	Heart failure regressed
11	M	35	Chronic pyelonephritis	Congestive heart failure	4	LVH (3)	230/130	130/100	20	4	Good	Regression of malignant phase; Parkinsonism due to reserpine
12	F	30	Pregnancy toxaemia	Congestive heart failure; recurrent abortions	1	LVH (4)	220/135	140/110	10	3	Good	Pregnant 24/52, condition satisfactory
13	F	25	Pregnancy toxaemia	Recurrent left ventricular failure in pregnancy	1	LVH (4)	190/130	140/100	80	8	Poor	No further LVF but marked tolerance. Treatment abandoned
14	F	44	Chronic pyelonephritis	Toxaemia of pregnancy	1	LVH (1)	180/130	120/90	5	3	Good	Headache ceased
15	F	50	Essential	Headache, dizziness	1	N	180/125	130/80	5	3	Good	Headache ceased
16	M	20	Chronic pyelonephritis	Visual	4	LVH (3)	240/185	110/80	60	9	Poor	Regression of malignant phase. Tolerance; marked curare-like effects. Treatment abandoned
17	M	21	Chronic pyelonephritis	Congestive heart failure	4	LVH (3)	260/140	150/100	5	4	Good	Regression of malignant phase
18	M	52	Essential	Congestive heart failure	N	LVH (4)	190/140	140/110	10	5	Good	Heart failure regressed
19	F	24	Chronic glomerulonephritis	Toxaemia of pregnancy	N	LVH (1)	210/135	130/70	15	9	Good	Second pregnancy taken successfully to term
20	M	36	Chronic pyelonephritis	Congestive heart failure	1	LVH (4)	175/130	140/90	50	3	Good	Heart failure regressed
21	F	43	Chronic pyelonephritis	Congestive heart failure	2	LVH (3)	195/125	150/110	35	8	Good	Heart failure regressed. Patient also has endomyocardial fibrosis
22	F	25	Chronic pyelonephritis	Congestive heart failure, renal failure	4	LVH (3)	220/170	110/80	15	3	Good	Regression of malignant phase and renal failure. No recurrence of heart failure
23	F	35	Chronic pyelonephritis	Congestive heart failure, renal failure	4	LVH (4)	200/140	120/80	15	3	Good	Regression of malignant phase; heart failure controlled
24	M	24	Chronic pyelonephritis	Left ventricular failure	1	LVH (4)	200/110	120/80	10	3	Good	Regression of LVF

* On mecamylamine and reserpine.

LVF = left ventricular failure. LVH = left ventricular hypertrophy. N = normal. NT = not taken.

Chest X-ray: Moderate left ventricular enlargement. ECG: LVH (4).

Mecamylamine was started on 5 September and gradually increased to 10 mg. daily. He has been on this dose ever since. Within 2 months his papilloedema had gone and a recent examination revealed only grade-2 fundus changes. His headaches have been completely relieved and apart from slight puffiness around the eyes he has no symptoms. Monthly blood ureas since were 125, 71, 87, 56 mg. per 100 ml.

Comment. This was a case of gross uraemia at the time of recognition of a malignant phase and treatment has been successful in relieving it.

Table I shows, in addition to a summary of initial clinical data, basal blood-pressure levels both before treatment and after stabilization on mecamylamine.

In all cases reserpine was given with mecamylamine from the beginning, and therefore the results reported are based on the degree of control achieved with both agents. Toxic effects with the dose of reserpine used were unusual, apart from occasional nasal stuffiness and in one case (case 11), Parkinsonian tremors.

In 4 cases (4, 7, 13 and 16) a good result was not achieved; the reasons for the failure were considered to be as follows:

Case 13 rapidly developed tolerance to the hypotensive action of mecamylamine. The dose reached was the highest in the series, 80 mg., and in view of the extreme rapidity of development of tolerance it was considered unwise to increase her dose further. In cases 7 and 16, one (case 16) with malignant hypertension, tolerance was associated with disabling muscular weakness due to curare-like effects of mecamylamine (Stone *et al.*, 1956) which interfered with the patients' occupations. Case 4 developed mecamylamine tremor (Harington and Kincaid-Smith, 1958). His history was as follows:

Case 4, a male Ganda cultivator aged 49, was first seen in August 1957, when he was admitted in congestive heart failure.

On examination. Blood pressure 250/150 mm. Hg. Atrial gallop rhythm; left ventricular hypertrophy; fundi grade 1. Urine contained a trace of albumen. Blood urea 30 mg. per 100 ml.

Treatment. Benign essential hypertension having been diagnosed he was treated with oral pentolinium, to which reserpine 0·25 mg. twice daily was later added. This regime reduced his blood pressure, and his heart failure regressed.

Mecamylamine therapy. In May 1958 mecamylamine was substituted for the pentolinium, 50 mg. daily being necessary to keep the blood pressure down to 140/90 mm. Hg. with the patient in the erect position for the greater part of the day. This dosage, however, caused troublesome side-effects at first principally constipation and abdominal distention, dry mouth, and blurred vision, but later micturition difficulty as well.

Readmission. In September 1958 he developed tremor of the hands and 3 days later his condition deteriorated. On readmission on 29 September he had a coarse generalized shaking affecting the whole body, present at rest and accentuated on attempting voluntary movement. His speech was slurred and jerky. There was a general increase of muscular tone, and in the deep reflexes the plantars remained flexor. His mental state, however, was clear, but he was extremely restless, nervous and agitated. His mouth was extremely dry. Blood urea was 27 mg. per 100 ml. This striking clinical picture persisted for over a week and gradually subsided with the withdrawal of mecamylamine. By early October, 10 days after mecamylamine had been stopped, his condition had returned to normal. During this period he had been treated with pempidine because his blood pressure had risen after the cessation of hypotensive treatment. He was discharged from hospital on 10 October 1958 on a combination of pempidine and reserpine and when last seen had remained well.

Comment. This patient undoubtedly suffered from a syndrome well recognized as an occasional complication of treatment with mecamylamine.

In summary we consider that treatment was ineffective in 4 of the 24 patients because of undesirable side-effects attributable to mecamylamine.

Side-effects after Administration of Mecamylamine

The side-effects discussed here are those which were due to mecamylamine alone. Doubtless, the use of reserpine with the mecamylamine enabled a smaller mecamylamine dosage to be given to obtain the same hypotensive effects. Thus fewer side-effects were evident than would have been the case with the use of mecamylamine alone. All the side-effects which may follow the administration of a ganglion-blocking drug occurred in the series. The side-effects observed (Table II) were as follows:

TABLE II. SIDE-EFFECTS OF MECAMYLAMINE IN 24 PATIENTS

		No. of cases
Alimentary	Dry Mouth	6
	Constipation and abdominal distension	19
	Diarrhoea	1
Genito-urinary	Dysuria	2
	Impotence	2*
Neurological	Blurred vision	7†
	Fatigability	4
	Tremor	1

* Present before treatment in one case.

† One case of secondary optic atrophy following malignant phase.

Dryness of the mouth was not common and was severe in only 2 cases.

Constipation was often troublesome and required constant vigilance. A regular aperient was usually prescribed and patients were encouraged to take whichever aperient suited them best, e.g. vegetable laxative tablets, salts, liquid paraffin. None of our patients on treatment developed paralytic ileus. Distention was frequently associated with constipation but did not seem to be particularly distressing.

Diarrhoea. Contrary to the experience of Kitchin *et al.* (1957) only one patient (case 16) in this series developed diarrhoea. The explanation may lie in the smaller doses required for stabilization of our cases, or there may be a dietetic factor. The staple diet of most of our patients is cooked unripe banana, which is a bulky item of diet and its digestion is probably slow.

Dysuria. Difficulty in micturition developed in 2 patients but was not serious.

Impotence. In 2 patients impotence was of sufficient degree for the complaint to be raised spontaneously. In one, however, the complaint had been present before treatment with mecamylamine had been begun. No patients were questioned specifically on this side-effect.

Blurring of vision from impairment of accommodation was complained of by 7 patients. Many of our patients were illiterate and perhaps might have complained more had they been great readers. One patient, a nun (case 3), benefited from a change of spectacles.

Fatigability. As in the experience of Smirk and McQueen (1957) and Kitchin *et al.* (1957), we also noted that some patients complained of a vague and general feeling of malaise and fatigue. These and other curare-like effects were so disabling to 2 patients that (as mentioned earlier) treatment had to be abandoned.

Mecamylamine tremor. Two patients developed tremor. In one only (case 4, see above) could it be ascribed to mecamylamine, on cessation of which the tremor disappeared. In the other case (case 11) tremor started 3 weeks after commencement of therapy, while the patient was on 20 mg. of mecamylamine and 0·5 mg. of reserpine daily. The tremor was of a Parkinsonian quality and characterized by 'pill-rolling', mask-like face, slowness of movements, and shuffling gait. The syndrome did not reverse when mecamylamine was withdrawn and as it persisted as long as reserpine was continued, it was probably due to the reserpine. A similar syndrome has been described, as due to reserpine, by Kline (1954) and Stead and Wing (1955).

SUMMARY

29 African patients with severe hypertensive disease were considered suitable for long-term treatment with ganglion-blocking drugs. Treatment regime consisted of mecamylamine in combination with reserpine. 24 cases were followed up for periods up to 9 months.

The majority of patients were cases of renal rather than essential hypertension.

The results were good or excellent in 20 cases and poor in 4 cases. Failure to achieve a good result was due to resistance to mecamylamine and intolerance of side-effects. Side-effects occurred in most cases but were rarely disabling. Constipation and abdominal distention were the most frequent side-effects.

Our thanks are due to Prof. A. W. Williams for inspiration and encouragement, to the physicians at Mulago Hospital for kindly referring cases for investigation and treatment, and to our patients for their good humour, charm and forbearance.

We are indebted to the Director of Medical Services, Uganda Administration, for facilities at Mulago Hospital, and to Messrs. Merck, Sharp and Dohme International for an initial supply of mecamylamine. Generous research grants from Makerere College and the Director of Medical Services, Uganda Administration, enabled the purchase of drugs for maintenance therapy.

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JUVENILE HYPOTHYROIDISM

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'Juvenile hypothyroidism' is a generic title for a group of diseases arising from a variety of causative factors and characterized by subthyroidism with apparent onset in childhood. The term 'cretinism' in this paper is reserved for those cases which show gross hypothyroidism with signs

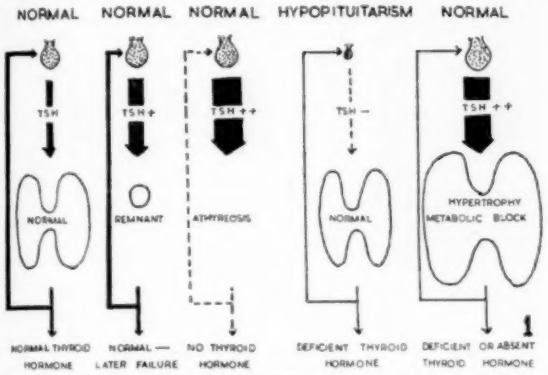

Fig. 1. Simple diagrammatic representation of aetiological factors in hypothyroidism.

of mental deficiency and with onset at birth, or very soon after. Because of the variety of types and of aetiological factors (Fig. 1)—often unknown—it might be better to refer to the cases as belonging to the broad group comprising juvenile hypothyroid syndromes.

Five cases are presented, with a brief functional classification of hypothyroidism in childhood and adolescence, and a discussion on differentiating features.

CASE REPORTS

Case 1. European female aged 14 years

This patient was referred for investigation by her mother because of failure to grow. Pregnancy and delivery had been normal and she had passed all her milestones at the expected times. She started school at the age of 7 and made good progress; in fact, the year before attending at hospital had come top of her class. However, from about the age when she first attended school it became gradually apparent that she was not growing normally and, in addition, she often complained of feeling cold.

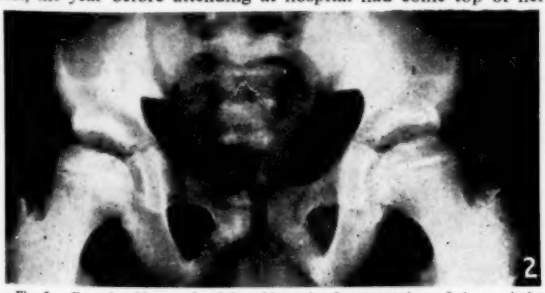

Fig. 2. Case 1. X-ray of pelvis. Note the fragmentation of the capital femoral epiphyses and of the epiphyses of the trochanters.

On examination, she was found to be a quiet, short (52 inches), obese child with a dry, scaly skin. There was no evidence of secondary sexual development. The sleeping pulse rate was 60 beats per minute and her thyroid was not palpable. The rest of the physical examination was normal. Her bone age was estimated to be 13 years (Fig. 2).

The electrocardiograph (ECG) showed low voltage QRS complexes under 10 mm. in leads V4-V6, and flattened T-waves (Fig. 3). The serum cholesterol was 636 mg.%. The basal metabolic rate BMR was −52%.

Treatment was begun with 10 μg. of tri-iodothyronine twice daily for 7 days and then continued with 0·1 mg. of L-thyroxine twice a day. Within a week the ECG showed increased voltage

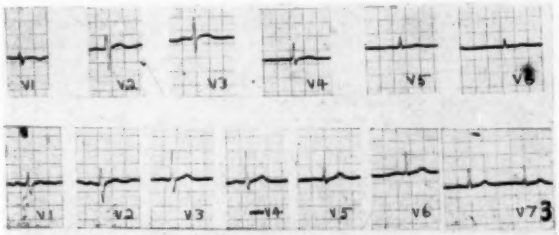

Fig. 3. Case 1. Upper tracings = ECG taken on admission. Lower tracings = ECG taken after treatment with tri-iodothyronine for 1 week. Note the increasing voltage of the T-waves in the left praecordial leads.

and higher T-waves (Fig. 3) and there was marked subjective improvement. Four months later she had grown 2 inches, her breasts had begun to develop (Figs. 4A and 4B), the sleeping pulse rate was now 84 beats per minute, and the BMR was −3%.

Case 2. Coloured male aged 18 years

This patient presented himself at medical out-patients with the complaints that he was too short and was much slower than his friends of the same age. He had had no schooling and was unable to give a detailed history or produce any reliable witnesses. He said he always felt cold.

On examination he lay in bed completely covered by blankets on a warm day. He was short (53 inches with a span of 50 inches) and obese and had a thick, dry skin; coarse facial features (Figs. 5A and 5B). His pulse rate at rest was 46 beats per minute. There was a nodular goitre affecting mostly the right lobe and the isthmus. The rest of his physical examination, including secondary sex characters, was normal.

It was not possible to obtain an accurate estimation of his intelligence but, taking into account his social background and lack of formal schooling, he appeared to be of low normal intelligence.

His bone age was estimated to be 13-15 years.

The ECG was within normal limits. The serum cholesterol was 269 mg.%. The BMR was −30%.

Treatment was begun with 10 μg. of tri-iodothyronine twice daily and within a week it became apparent that there was improvement. He no longer lay in bed curled up beneath the blankets; he was active, alert and helpful about the ward. His resting pulse rate had risen to between 80 and 90 beats per minute. He was discharged on a maintenance dose of 0·1 mg. of L-thyroxine twice daily.

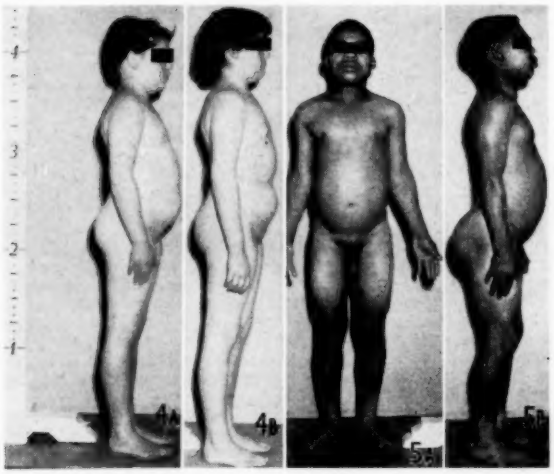

Fig. 4*A*. Case 1. Before treatment.
Fig. 4*B*. Case 1. After treatment for 4 months with thyroid hormone. Note change in profile.
Figs. 5*A and* 5*B*. Case 2.

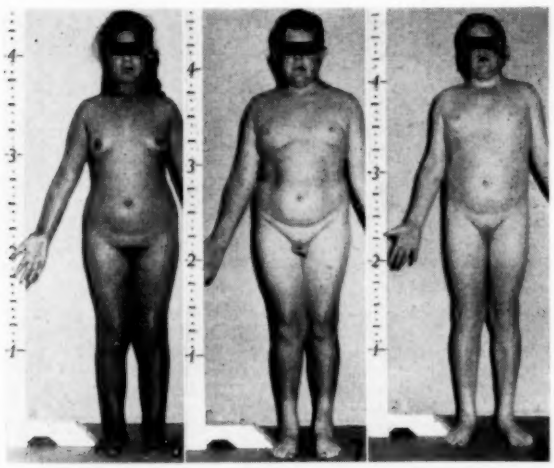

Fig. 6. Case 3. *Fig.* 7. Case 4. *Fig.* 8. Case 5.

Case 3. Coloured female aged 19 years

This patient attended at medical out-patients complaining of shortness of stature, of a swelling in the neck which had been present about 5 years, and of increasing obesity. Her mother stated that her milestones, following a normal pregnancy and delivery, had all been normal. When she first attended school at the age of 6 her height had been normal for her age. She was able to read at 7 years. Dating from this time she failed to grow and her performance at school deteriorated. She eventually passed standard 4 at 14 years and then left school. The rest of her family were all normal, except for her mother, who had a multinodular non-toxic goitre. She had begun to menstruate only 1 month before attending at hospital.

On examination she was found to be short (51 inches) and obese (Fig. 6), with a thick, dry skin and a hoarse voice. There was no body hair. The breast development was fair, but on palpation seemed to be mainly fat. There was a large cystic mass in the upper pole of the right lobe of the thyroid.

Her bone age was estimated to be 13-14 years.

The ECG was within normal limits. The serum cholesterol was 325 mg.% before treatment.

She was treated as an out-patient with 0·1 mg. of L-thyroxine twice a day and made steady progress. She became brighter, lost weight and had more energy. The thyroid swelling gradually decreased and eventually disappeared within 3 months. One year later she had grown 1½ inches, but her voice was still hoarse and she still had no pubic hair, although her periods were now lasting 4 days instead of 1 day, and she showed good development of her nipples.

Fig. 9. Case 4. X-ray of spine. Note the absence of secondary ossification centres.

Case 4. European male aged 15 years

The complaints in this case were of mental dullness and failure to grow. The patient weighed 10½ lb. at birth, after a normal pregnancy and delivery. His mother stated that he passed all his milestones at the expected times and by 2 years was walking and talking normally. From this time, however, he made no further progress and at the age of 3 years was started on 'thyroid'. No illness of any sort had been noted and no swelling in the neck had been observed. For a period of 1 year before his visit to hospital he had not had any thyroid preparation. The rest of his family were all normal and tall.

On examination he was found to be short (55 inches) and obese (Fig. 7), with many brown moles. The skin was dry and tended to crack. There was no secondary sexual development. The rest of the physical development was normal and the thyroid was not palpable. His bone age was estimated at 9 years (Figs. 9 and 10).

The ECG showed bradycardia and flattened T-waves. The serum cholesterol was 425 mg.%.

He was treated as an out-patient with 0·1 mg. of L-thyroxine twice daily. When examined on subsequent visits he was much more alert and active, and an ECG done later was now within normal limits.

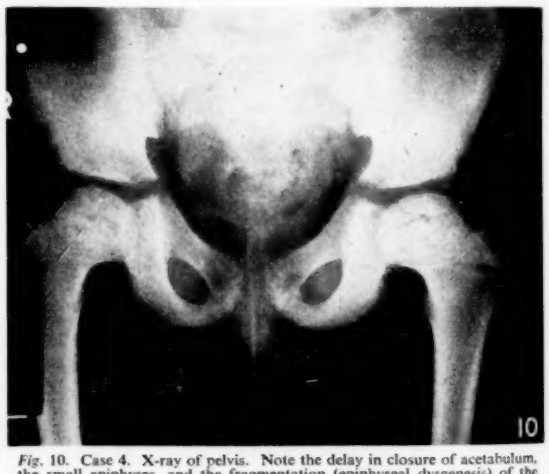

Fig. 10. Case 4. X-ray of pelvis. Note the delay in closure of acetabulum, the small epiphyses, and the fragmentation (epiphyseal dysgenesis) of the epiphyses of the lesser trochanters and of the capital femoral epiphyses.

Case 5. European male aged 17 years

In 1950 at the age of 9 years this patient underwent a craniotomy for a craniopharyngioma. He had developed normally in physique and intelligence at this stage. The operation was successful and he was able to lead a normal life, except that he failed to grow, developed no secondary sex characteristics, and suffered from polydipsia and polyuria.

On examination he was found to be a bright and cooperative young boy who measured 57 inches in height. He was obese

and there was complete absence of secondary sex development (Fig. 8). His resting pulse rate was 90 beats per minute. There was bitemporal hemianopsia and the optic discs were atrophic. The thyroid was normally palpable and the rest of the physical examination was essentially normal. Skull X-ray showed the typical suprasellar calcification of a craniopharyngioma.

His bone age was estimated to be between 13 and 14 years.

The ECG was within normal limits. The serum cholesterol was 230 mg.%. The BMR was −22%. The protein-bound iodine (PBI) was 2·6 μg. per 100 ml. (normal range 4-8 μg. per 100 ml.).

Treatment was begun with 0·1 mg. of L-thyroxine twice daily, together with 5 mg. of prednisone daily and pitressin tannate as required.

SUMMARY OF THE 5 CASES

	Case 1	*Case 2*	*Case 3*	*Case 4*	*Case 5*
Age (yrs.)	14	18	19	15	17
History of previous development	Normal up to ± 7 yrs.	Not available	Normal up to ± 6 yrs.	Normal up to 2 yrs.	Normal up to 9 yrs.
Epiphyseal dysgenesis	Yes	Yes	No	Yes	No
Height (inches)	52	53	51	55	57
Obese	Yes	Yes	Yes	Yes	Yes
Secondary sex development	No	Normal	Late	No	No
Thyroid	Not palpable	Goitre	Nodule	Not palpable	? Normal
ECG	Abnormal	Normal	Normal	Abnormal	Normal
BMR	−52%	−30%	—	—	−22%
Serum cholesterol before treatment	mg.% 636	mg.% 269	mg.% 325	mg.% 425	mg.% 230
PBI	—	—	—	—	2·6μg.%
Bone age (yrs.)	13	13-15	13-14	9	13-14
Aetiology	Acquired ? thyroid atrophy	Non-endemic goitrous hypothyroidism	Partial athyreosis (mid-line remnant)	Acquired ? thyroid atrophy	Secondary to hypopituitarism

CLASSIFICATION OF SUBTHYROIDISM IN EARLY LIFE

The division is broadly congenital hypothyroidism and acquired hypothyroidism; there may be some overlapping of aetiological factors.

Early (congenital) Hypothyroidism

1. *Anatomic Dysgenesis*

There is in this category an embryonic defect in the development of the thyroid, which may be completely absent (athyreosis) or may be represented by small functioning remnants in the neck or attached to the tongue. It has been shown that these 'remnants' are hyperfunctioning.[1] In the latter case the infant may be normal at birth and remain so for a period until the overstressed remnant atrophies and the child becomes hypothyroid. In the former case the condition of hypothyroidism with cretinism will certainly be manifest unless treatment is begun soon. Often despite timely and adequate treatment the infant will be mentally defective with good physical development. It is thought that the lack of development of the brain may begin *in utero*, although Stanbury and Querido[2] suggest that there may be an associated mental defect in some of these cases.

The foetal thyroid becomes a functioning structure by the 12th week of gestation and supplies sufficient thyroid hormone for the needs of the foetus.[3,4] This fact, together with the study of retarded skeletal development in the newborn, enables an estimate to be made of the time of onset of intra-uterine failure of thyroid hormone.

It is estimated that 45% of athyreotic babies adequately treated before the age of 6 months have attained the range of normality in mental development. The prognosis in individual cases is difficult and variable.

2. *Nutritional Hypothyroidism* (*endemic goitrous hypothyroidism or cretinism*)

This disease occurs in environments where there is lack of iodine. Well-known areas are the Alps and the Himalayas.

A typical case occurs in a family with progressively severe goitres, producing finally a goitrous, mentally deficient infant. The thyroid may undergo atrophy early. It is thought by some observers that these babies are functionally athyreotic, thus explaining the high incidence of mental deficiency or cretinism. Stanbury and Querido[2] suggest that a genetically inborn error of metabolism may be accentuated by iodine lack in the environment (see below). This would help to explain why not all infants are affected despite widespread iodine lack. A high uptake of ^{131}I shows the avidity of the thyroid for iodine. Urine ^{127}I is low.

3. *Inborn Errors of Metabolism* (*non-endemic familial goitrous hypothyroidism*)

The synthesis of thyroid hormone has been subject to detailed study. It is a complex process involving several enzyme systems in successive steps leading to the final principles. Failure of an enzyme system at any point in the chain can and does occur, with resultant deficient thyroid hormone.

Three main types of familial hypothyroidism, as follows, have been studied, characterized by a specific enzyme failure at a particular stage. All the patients, either early or in later years, have an enlarged hyperplastic thyroid gland.

(*a*) *Failure in the organification of iodide.* The gland may take up iodine but is unable to oxidize it to elemental iodine in the absence of an oxidase which is normally present.[5]

(*b*) *A postulated failure of coupling of iodotyrosines.* Thyroxine is formed when 2 molecules of di-iodothyronine are coupled; 3-3′-di-iodothyronine is formed by coupling of 2 molecules of mono-iodotyrosine; and so forth. Cases have been described with large goitres and mental retardation and with large amounts of mono- and di-iodotyrosine in the thyroid but only low peripheral concentration of thyroxine.[6]

(*c*) *Lack of de-iodination of iodotyrosines.* Thyroglobulin normally undergoes proteolysis with the release of thyroxine, tri-iodothyronine and mono- and di-iodotyrosine. The last two substances are de-iodinated by a specific enzyme in the thyroid[7] and do not appear in the blood.

Three cases of goitrous hypothyroidism have been described that were unable to de-iodinate di-iodotyrosine. Two were cretins, one was normal. When these cases were given intravenous injections of labelled di-iodotyrosine it appeared unchanged in the urine, whereas in normal people it did not appear in the urine.[7]

The causative factor here seems to be a loss in hormone precursors. The familial incidence in this type is high.

4. *Congenital Goitres with Hypothyroidism*

In babies whose mothers have received drugs such as thio-uracil, iodides, etc., goitres may be present at birth, as well as cretinism. After birth the goitres usually disappear and the infants may be toxic, euthyroid or hypothyroid.[8-10] Retarded osseous development and epiphyseal dysgenesis may provide evidence of intra-uterine hypothyroidism.

There remain some cases in this group in which the aetiology is obscure. The infants who are hypothyroid at birth may remain so and should be treated if this occurs.

Later (acquired or minor congenital) Hypothyroidism

(In these cases there must be evidence of preceding normal thyroid function.)

1. *Juvenile Hypothyroidism* (*'Juvenile myxoedema'*)

There are a number of causative factors, some of which have already been discussed, viz. partial athyreosis, infective or infiltrative diseases, and thyroiditis, including Hashimoto's disease in children, or primary failure of the thyroid.

The term 'juvenile myxoedema' should be reserved for those cases which in addition have the typical skin changes as in their adult counterpart. Often these patients are of normal intelligence and may not even appear as sluggish as the adult with myxoedema.

The onset of the disease is characteristically insidious, with gradual slowing of growth and activity followed at a later stage by a more abrupt failure of growth.

The thyroid stimulating hormone (TSH) levels are reported as being high in this group.[11]

2. *Secondary Hypothyroidism*

This group comprises cases which have inadequate thyroid function secondary to pituitary failure, either as a specific failure of TSH or as part of the syndrome of panhypopituitarism. In these cases it may be demonstrated that the TSH levels are low and that the thyroid is capable of responding to TSH stimulation unless the gland has become severely involved. Severe involution is seldom present and repeated stimulation by exogenous TSH will usually produce adequate thyroid response.

The diagnosis may present some difficulties, because the symptoms are usually mild and the skin changes of juvenile myxoedema absent. The main problem is to differentiate the disease from dwarfing due to deficiency of growth hormone or hypopituitarism, for the two conditions may coexist.

Wilkins[12] points out that in dwarfing due to hypothyroidism the skeletal proportions are infantile, whereas in dwarfing due to lack of growth hormone the proportions are adult.

As in the previous categories the bone age is retarded, and this can be accelerated by the exhibition of thyroid. Thyroid will not increase the stature in deficiency of growth hormone.

The PBI estimation is useful; the level of this is always low, provided the usual precautions are taken.

Finally radio-active iodine studies may be utilized, particularly in combination with TSH stimulation, to establish the existence of normal potential thyroid function.

DISCUSSION

Wilkins[15] states that hypothyroidism is one of the commonest of all endocrine disorders of childhood and adolescence in the United States of America. There is a wide range in the spectrum of the disease, the clinical manifestations of which are directly proportional to the amount of thyroid hormone produced and inversely proportional to the age of onset of thyroid dysfunction. At the one end of the scale there is the floridly myxoedematous cretin—cold, sluggish, with coarse facial features, and markedly stunted. At the other end of the scale there is the mildly hypothyroid patient whose main complaint is of failure to grow satisfactorily.

Diagnostic Features

The gross examples of the disease are adequately described in the text-books and need no further amplification. Difficulty may arise with the mild cases and for this reason a few points are worth emphasizing.

Bone changes. Most observers lay considerable stress on retarded osseous development. The skeletal system in children is most sensitive to thyroid deficiency. The bone age is always retarded unless thyroid failure is of recent onset.[13,14] Delay in the appearances of epiphyseal centres, primary and secondary, plus irregular ossification of the epiphyses—epiphyseal dysgenesis—which gives rise to an irregular stippling in the radiograph, is practically pathognomonic of hypothyroidism.[12,15] This is not a reported feature of secondary hypothyroidism. However, the radiological appearance of osteochondritis deformans is sometimes very similar, although in it there is no evidence of retarded osseous development. The hip joints, the epiphyses of the knee, the wrist joints and the femoral trochanters may be affected. Jackson *et al.*[16] drew attention to the vertebral bodies, which are 'unusually small, square and dense, with irregular outlines'. They also point out that occasionally one vertebra may be particularly small, wedge-shaped and displaced backwards. The latter features are also seen in gargoylism. The failure of the appearance of the vertebral secondary ossification centres is partly responsible for the short stature.

The serum cholesterol is usually high in the untreated cases except in the secondary hypothyroidism of hypopituitarism, when it may be normal.

Electrocardiograph. The ECG changes are very valuable, both in the diagnosis and in the evaluation of the efficacy of therapy. The usual changes are a prolonged P-R interval and low voltage QRS complexes and flattened T-waves. Bradycardia is frequently mentioned as an additional feature, and in this respect it is interesting to note the two cases of adult cretins reported by Jackson[17] with pulse rates above normal. Benda and Falta[18] state that tachycardia is a recognized occasional feature of infantile cretinism.

Sexual features. The development of secondary sexual features is usually delayed, but not necessarily so.[17] Case 2 had normal development of sex characteristics, with a large penis. Jackson[17] states that in male adult cretins the penis and testicles may be outsize. Case 3 menstruated and, while on treatment, the duration of her menses increased from 1 day to 4.

The circulating thyroid hormone was estimated indirectly by measuring the PBI, which is always low in hypothyroidism. It must be remembered that the administration of iodides *in any form* may result in high values. The normal range is 4-8 μg.%. Case 5 had a PBI of 2·6 μg. % and with the

exception of the skeletal changes this was the best objective evidence of hypothyroidism.

Basal metabolic rate. The BMR may be misleading, although in most cases of hypothyroidism it is singularly low.

Radio-active iodine studies are used to establish the presence—or absence—of functioning thyroid tissue by direct scanning methods over the neck. In addition, the metabolic turnover and synthesis of the thyroid hormones can be investigated. In nutritional goitrous hypothyroidism most cases will take up ^{131}I quite rapidly with a subsequent slow rise in the protein-bound ^{131}I, whereas in the non-endemic goitrous hypothyroid patients (metabolic disorders) ^{131}I may be taken up quite avidly in some cases, but because they cannot manufacture thyroid hormones adequately there will be no significant rise in the protein-bound ^{131}I.

Therapy and response. The response to therapy may be manifested by increased activity, by increased growth, and by ECG and metabolic changes. In doubtful cases therapy may be withheld for a period in order to ascertain whether symptoms recur and biochemical changes regress.

The two commonly used drugs are L-thyroxine and tri-iodothyronine. The latter is 5 times as antigoitrogenic as thyroxine. Desiccated thyroid extract is not frequently used in this hospital because the results from it are variable owing to its unpredictable potency. Intravenous thyroxine produces a maximum response in the BMR within 7-10 days, whereas tri-iodothyronine provokes a maximum response in a matter of 24 hours. Both preparations produce a rapid and marked drop in the serum cholesterol.[19] Cessation of therapy may result in a return of the cholesterol to or beyond its former high level in 6 weeks.[12]

Conclusions from Present Cases

The patients described are presented here as examples of juvenile hypothyroidism. All the patients except case 2 gave positive histories of previous normal development up to a certain age. Case 2 had no witness to testify to early normal development, but one could infer from clinical examination and assessment of his ability to cope with his environment that his mental retardment was not great. In other words, one presumes on reasonable grounds that his thyroid production, although inadequate, was sufficient for him to develop, albeit sluggishly.

Even with the aid of modern laboratory methods, the precise aetiology of the disease in individual cases may remain unknown; sometimes it is quite obvious, as, for example, in case 5, who had suffered pituitary destruction by a craniopharyngioma. Case 3 had had a thyroid nodule in the neck which rapidly regressed on thyroid treatment; it is logical to conclude that she is probably an example of partial athyreosis, particularly because of the subsequent progress and behaviour of the nodule, the exogenous thyroid 'damping' the pituitary and allowing the stressed and hypertrophied remnant to subside.

When there is no thyroid tissue palpable, as in cases 1 and 4, the problem is more difficult. If there is no history of previous goitre or of thyroiditis or operation, the aetiology may not be discovered.

Radio-active iodine studies may or may not be helpful.

SUMMARY

Five cases of juvenile hypothyroidism are presented, together with classification and discussion of multiple causative factors.

I wish to thank Dr. W. P. U. Jackson for much encouragement and advice in preparing this paper. I am also indebted to Prof. J. F. Brock, Prof. F. Forman, Dr. R. Hoffenberg, and Dr. C. P. Dancaster for helpful criticism and advice. Dr. J. G. Burger, Medical Superintendent of Groote Schuur Hospital, kindly gave permission for publication of the case records of the patients concerned. I am grateful to Mr. B. Todt for the photographs.

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THE MEDICAL PRACTITIONER AND WORKMEN'S COMPENSATION

J. P. GROBBELAAR *Workmen's Compensation Commissioner, Pretoria*

An article by Neil Carter, under the heading 'General characteristics of hearing procedures', was published in *Compensation Medicine* (**10**, 14, September 1958), the official publication of the American Academy of Compensation Medicine Inc., New York, USA. This article deals with problems relating to the hearing and adjudication of compensation claims by members of the staff of the New York State Workmen's Compensation Board and, as can be expected, contains a reference to the inevitable *medical aspect* without which the 'workmen's compensation' picture cannot be complete.

A great deal of what the author of this article says regarding the medical aspect of workmen's compensation is relevant to this country for, in our experience, there are many medical practitioners who do not appear to realize the importance of their status in workmen's compensation administration, and the impact which their individual contributions may have on the daily application of an intricate piece of legislation involving so many aspects of human nature and its attendant weaknesses.

The author refers, for instance, to the tendency on the part of some doctors to assume the role of advocate, whereas they should confine themselves strictly to technical fact-finding and unbiassed professional opinion. I think that in most cases in South Africa this is done unwittingly. A doctor may be misled by the woeful tale of his patient and be ensnared by sympathy or a desire to give the patient some satisfaction.

We should, however, always be on our guard. Although it is our duty to concede whatever is legitimately due to the claimant, it must, nevertheless, continually be borne in mind that workmen's compensation patients are not always like ordinary patients for the simple reason that they are often inclined to view their malady, and its cure, against a background of possible compensation. According to his mental make-up, and the absence or presence of

old-standing ailments, such a patient may or may not be a willing subject for attempt at optimum physical restoration.

Doctors should, therefore, exercise meticulous care in formulating diagnoses and expressing opinions to, or in the presence of, a patient, particularly a patient with a 'history'. The stock phrase 'aggravation of a pre-existing condition' is too often used lightly and may lead to far-reaching results, detrimental both to the patient and to the insurance carrier.

Too often we on the administrative side, as 'referees' are placed in a difficult position because of a loose diagnosis or an exaggerated claim made by a specialist or general practitioner on behalf of a compensation claimant. I refer particularly to those 'dead beats' and cases where 'legal mortis' (to use Mr. Carter's phraseology) has set in; those never-to-be-satisfied cases who want to be compensated for all their inherent ills and weaknesses and who will tramp from one doctor to another in the hope of finding someone who will support their cause.

Once morbid ideas, sometimes based on mere conjecture, have been put into the mind of such a patient—be it a matter of connecting some obscure complaint or symptoms of a dubious character with an *injury at work*, or of suggesting an inflated degree of disablement, the difficulty of resisting unjustifiable demands arising from these ideas, and of undoing the psychological damage done, can be well imagined. Quite often this situation leads to complaints to a Minister of State, Member of Parliament, Trade Union official, or other influential lay person who, quite naturally, is at a loss to understand why 'the word of the workman's own doctor' is not accepted.

In the interests of all concerned *fair judgment* should be *first* on the list of ethical rules when handling workmen's compensation cases, whether for treatment or for examination and report, or when giving evidence at a formal hearing in a disputed case. This matter, in my opinion, is of great importance and deserving of the earnest attention of the medical profession.

Abstract of Article 'General Characteristics of Hearing Procedures'

Every doctor on a compensation panel is vitally concerned with the law. Under the law medical treatment is regarded as an essential part of the compensation system. However, the question that must still be answered is why it costs so much to settle medical fees in compensation cases in this State? The accusation has been made that over-treatment is a common practice; and it has been stated that medical precedent, of a disturbing nature, is being set by so many claims being tabled for treatment of back pains, headaches and other types of intangible ailments which resist positive diagnosis and recovery.

The doctor, in relation to hearing procedures, can be of assistance by filing prompt and complete reports. The doctor has a responsibility, not only in taking care of compensation patients, but also in recording and reporting histories in an accurate manner, and in exercising care in reporting an opinion of causal relationship. We see too many cases where doctors fail to report, for example, histories of pre-existing injuries or disease involving the same pathology as in the compensation case.

We also see too many doctors participating in compensation hearings as advocates and not as doctors. We believe that a doctor can best serve his profession, his patient, the community, and the law by not appearing as an advocate for one side against the other.

We all know that the initial history is generally the most accurate and truthful history. This is true since it is taken before outside influences enter into the case. I recently read a letter written by a doctor to a medical journal in which he described these extraneous influences as 'legal mortis'.

Once 'legal mortis' enters the case, symptoms arise which are so bizarre as to defy medical description, since the patient's desire to regain health comes in conflict with his desire to maintain compensation status until his case can be settled.

Would it not be helpful if the doctor reported when delayed histories were received; when, and in association with what circumstances, the change in story occurred; and when new symptoms were presented?

The doctor could also be helpful in reporting pathology and not complaints and in using diagnostic terms to describe injuries instead of using broad general terms such as 'low back derangement' or 'discogenic pathology'. Too often we have seen medical reports filed on first examination and within 24 to 48 hours, reporting pathology of an extremely serious nature, such as a herniated disc, as if the reporting of serious pathology will excuse the heavy bill for treatment which subsequently will be filed.

Too many doctors use vague terminology as a cover under which they can manœuvre. This type of doctor knows that claimants and their attorneys can profit from the use of non-specific diagnostic terminology.

If a doctor has to testify in a case where a definite diagnosis cannot be made and no objective findings are present to account for the subjective complaints, particularly after repeated negative examinations of the patient, it is the doctor's duty to disclose this fact.

Any discussion of hearing procedures must include the role of the referee. The referees have all the powers of the Workmen's Compensation Board with respect to the hearing and determination of claims for compensation, subject to an appeal or review by the Board. The judgment they exercise influences the entire trend of the law.

It is the duty of the referee to use his judgment in determining the issues in a case. Our sympathy must go out to the referee who, in attempting to fulfil his duties, encounters the belligerent type of claimant—the claimant who feels that all the presumptions of the law must be resolved in his favour. The job of the referee is not made easy when he is called upon to distinguish between honest claims and exaggerated claims.

The 'dead beats' and pension seekers in compensation, by involving calendars with the prolonged litigation their type of case produces, actually do most harm to the workman who is really hurt at work. The trial calendar that is crowded with fringe claims excludes the legitimate cases.

Each one of us should be interested in the future of compensation because it is our future too. No one wants to turn back the clock in this great social advance. The aim to improve the system is one in which we can all join.

QUESTIONS ANSWERED : ANTWOORDE OP VRAE

MENIÈRE'S DISEASE

Q—*I have a male patient aged 52 who has been subject to Menière's disease for some years. The attacks of vertigo, tinnitis and distortion of hearing have become extremely troublesome. Symptomatic drug treatment has been unsuccessful. Would you please outline the treatment of this condition. What is the place of surgical interference, e.g. destruction of the labyrinth, etc. in the treatment of this condition?*

A—The treatment of Menière's disease starts with making certain that the patient understands the nature of the condition so that his anxiety may be allayed and so that he may better adjust himself to the disadvantages of the disorder. With regard to the drug treatment of the disease it seems probable that phenobarbitone still provides one of the best long-term prophylactics. Traditionally, a search is made for septic foci and for allergic factors, and antihistamine injections may bring a temporary relief to a series of disconcerting attacks. Antihistamines are often used in the long-term therapy of these cases, avomine 25 mg. three times daily being commonly used. Stemetil 5–10 mg. three times a day may be found useful. Fluids should be restricted to 2½ pints per day and it may be worth while trying the patient on a low-salt diet, although this is a very difficult diet to maintain.

In cases where conservative treatment has failed to give adequate relief the question of surgery may have to be considered. Attacks on the stellate ganglion have not been successful in the hands of most workers. In unilateral cases destruction of the affected labyrinth with alcohol may be considered but one must keep in mind that the other ear may be affected later and that the ensuing deafness is a considerable disadvantage. In patients with only one labyrinth affected, Cawthorne prefers to dissect a piece of the membranous external canal. It is possible to cut the vestibular nerves

bilaterally and to leave the cochlear nerves intact, but the operation is one of great delicacy and difficulty.

The disadvantage of most surgical procedures is that hearing is lost and, in a bilateral case, this may be disastrous. Where only one vestibular labyrinth is affected surgery may be considered, but the value of surgery is reflected in the fact that there is no universally accepted procedure.

PARKINSONISM

Q—*Would you please enumerate the most effective drugs in current use for the symptomatic treatment of parkinsonism, indicating* (1) *the order of preference, and* (2) *a suggested scheme for treatment?*

A—The question of choice of drugs in parkinsonism is an individual one. For a mild case one of the solanaceous drugs is probably most suitable because of the low toxicity of this group of drugs compared with the more potent but more toxic piperidyl group.

A suggested scheme of treatment would be to start with hyoscine hydrochloride, gr. 1/200 three times daily, and to increase this dose from time to time up to the limit of tolerance (dry mouth and blurred vision) so that the patient might soon be having gr. 1/50 of the drug 3 times daily. If drowsiness is a concomitant, dexedrine, 2½ gr. should be added in the morning. If this is not satisfactory, one might try 2 mg. of artane 3 times daily, once more increasing the dose as necessary, since tolerance to all anti-parkinsonian drugs is soon developed. One of the antihistamines, such as thephorin (25 mg. 2 or 3 times daily) may be added to any of the anti-parkinsonian medications to increase their effectiveness. Finally, one might try disipal, 50 mg. three times daily, alone or in combination with another anti-parkinsonian drug. Disipal has the advantage of giving a sense of well-being and for that reason is a useful adjuvant to use at night since it may allow the patient to feel less anxious about sleep and the problems which so often enlarge as night falls. One might keep in mind the possibility of the operation of pallidotomy on youngish or rapidly advancing cases, particularly when the signs are largely unilateral.

The order of preference of the drugs is another individual matter. My own preference is reflected in the order in which the above drugs are discussed. There are very many other drugs on the market, but no great advantage is obtained by using endless analogues of the above preparations.

SOUTH AFRICAN MEDICAL AND DENTAL COUNCIL : SUID-AFRIKAANSE GENEESKUNDIGE EN TANDHEELKUNDIGE RAAD

The Minister of Health has approved an amendment to the rules of the South African Medical and Dental Council by the addition thereto of the following further qualifications under the heading:

(*a*) MEDICAL PRACTITIONERS:

Licensing Body	*Qualification*	*Abbreviation for Registration*
College of Physicians of South Africa	Diploma in Psychiatric Medicine	D.P.M.(C.P.S.A.)
Faculty of Anaesthetists of South Africa	Fellowship.	F.F.A.(S.A.)
University of McGill	Diploma in Public Health	D.P.H., Univ. McGill
University of Pretoria	Diploma in Public Health	D.P.H., Univ. Pret.
University of Stellenbosch	Master of Medicine (Pathology)	M.Med.(Path.), Univ. Stell.
University of Stellenbosch	Master of Medicine (Radiological Diagnosis)	M.Med.(Rad.D.), Univ. Stell.
University of Stellenbosch	Master of Medicine (Ophthalmology)	M.Med.(Ophth.), Univ. Stell.
University of Stellenbosch	Master of Medicine (Otorhinolaryngology)	M.Med.(L. et O.), Univ. Stell.

Die Minister van Gesondheid het sy goedkeuring geheg aan die wysiging van die reëls van die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad deur die toevoeging van die volgende verdere kwalifikasies onder die opskrif:

(*a*) GENEESHERE:

Lisensiëringsowerheid	*Kwalifikasie*	*Afkorting vir Registrasie*
Suid-Afrikaanse Kollege van Interniste	Diploma in Psigiatriese Medisyne	D.P.M.(K.I.S.A.)
Suid-Afrikaanse Fakulteit van Narkotiseurs	Lidmaatskap	L.F.N.(S.A.)
Universiteit van McGill	Diploma in Volksgesondheid	D.P.H., Univ. McGill
Universiteit van Pretoria	Diploma in Volksgesondheid	D.V.G., Univ. Pret.
Universiteit van Stellenbosch	Magister in Geneeskunde (Patologie)	M.Med.(Path.), Univ. Stell.
Universiteit van Stellenbosch	Magister in Geneeskunde (Radiologiese Diagnose)	M.Med.(Rad.D.), Univ. Stell.
Universiteit van Stellenbosch	Magister in Geneeskunde (Oogheelkunde)	M.Med.(Ophth.), Univ. Stell.
Universiteit van Stellenbosch	Magister in Geneeskunde (Oor-, Neus- en Keelheelkunde)	M.Med.(L. et O.), Univ. Stell.

PASSING EVENTS : IN DIE VERBYGAAN

The Italian Medical Association for Collaboration with the Press is organizing the first international congress of doctors who are concerned with the publishing of medical information, in Rome from 16 to 18 July 1959. The subject of the conference will be 'The importance at this moment of the social medical insurance problem in the world'. During the congress important problems concerning the publication of medical information will also be discussed and an attempt will be made to establish a principle for international collaboration. Practitioners who are interested in this subject and who might be in a position to attend this congress are invited to submit their names to the Secretary of the Medical Association at P.O. Box 643, Cape Town.

* * *

South African Society for Clinical Hypnosis. At an inaugural meeting held on Friday 20 March 1959, in Johannesburg, and attended by a representative section of the medical, dental, and psychological professions, a local Society for Clinical and Experimental Hypnosis was established. The meeting accepted as its main objectives the need to encourage cooperative relations among scientific disciplines with regard to professional research, discussion, and publication of all the manifold aspects of hypnosis. Dr. R. Geerling, a past-President of the Southern Transvaal Branch of the Medical Association of South Africa, proposed the election of a Steering Committee comprising members of the Medical, Dental, and Psychological Associations. The following members were elected: Dr. B. W. Levinsohn (medical), Dr. S. Rootenberg (dental), Dr. C. F. Kruger (psychological). This Committee was responsible for drawing up the Constitution which will be dealt with at the next meeting of the Society which will be held on 23 June. Further information may be obtained from the Hon. Secretary, Society for Clinical and Experimental Hypnosis, Department of Psychology, Tara Neuropsychiatric Hospital, P.O. Box 13, Saxonwold, Johannesburg.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

THIOCARBIN

The German firm of C. H. Boehringer Sohn announces the introduction of Thiocarbin and supplies the following information:

Thiocarbin is a new anti-seborrhoeic product for local application which is capable of superseding the various combinations of sulphur, salicylic acid and antibacterial substances hitherto used in the treatment of seborrhoeic conditions. This preparation possesses keratolytic and antibacterial properties, controls sebaceous secretion, and stimulates the reaction of the skin. Various types of seborrhoeic dermatitis, such as acne vulgaris, rosacea, and seborrhoeic eczema, have cleared up in a short time under treatment with Thiocarbin in strengths as low as 0.5% or less. Among over 3,000 patients successfully treated in clinical trials were many who had previously undergone a prolonged and ineffective course of treatment with the usual preparations. The new product constitutes an important advance in the radical treatment of these conditions, the social significance of which is of considerable importance.

For further information contact C. H. Boehringer Sohn, Ingelheim am Rhein, Germany.

BOOK REVIEWS : BOEKBESPREKINGS

PHYSIOTHERAPY OF VARICOSE ULCERS

The Physical Treatment of Varicose Ulcers. A practical manual for the physiotherapist and nurse. By R. Rowden Foote, F.I.C.S., M.R.C.S., L.R.C.P., D.R.C.O.G. With a section on *Electrical Adjuncts to Treatment* by Miss T. Wareham, M.C.S.P. Pp. xii+126. 88 illustrations. 15s. net + 1s. 1d. postage abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

This booklet is an attempt to provide British and other physiotherapists and nurses with a handy guide in their work in connection with the non-surgical treatment of varicose and postphlebitic ulcers of the lower third of the leg. This is a world-wide problem, causing not only much suffering but also, and more important even, much loss of man-power and great expense to the individuals concerned and to the state.

Whatever may be said to the contrary, it is very evident that the superficial type of ulcer is, in the last instance, due to incompetent communicating veins, which in turn are the result of too much *standing* and *too little exercise* of the muscles of the legs. All this is the result almost exclusively of our civilized way of living. The correctness of this view is supported by the type of treatment advocated by the author himself: Elevation, *strapping* and *massage*, supplemented by appropriate *exercises*.

The book is packed with useful information for the physiotherapist and nurse alike, and this is supplemented by many useful illustrations both of the two types of ulcer referred to and of the methods of massaging and strapping. There is nothing which cannot be clearly understood and put into practice by any intelligent nurse who takes the trouble to read the book carefully. It should be useful to many workers in this country.

G.C.A.v.d.W.

CUNNINGHAM'S ANATOMY

Cunningham's Manual of Practical Anatomy. 12th edition. Volume III. *Head and Neck: Brain*. Revised by James Couper Brash, M.C., M.A., M.D., D.Sc., LL.D., F.R.C.S.Ed., F.R.S.E. Pp. xii+520. 231 figures. 28s. net. London-New York-Toronto: Oxford University Press. 1958.

This volume follows the traditional pattern in being devoted to the dissection of the head, neck and brain. Nomenclature is based on the recommendations submitted to, and approved by, the Paris Congress of 1955. It is hoped that lasting agreement has been achieved in this respect so that the student will ultimately use the same terminology in the dissecting room and the wards. It is unfortunate that further modifications may be made in 1960.

There is little to disagree with in this excellent manual. I wonder if it is really necessary (p. 346) for the student to equip himself with 6 eyeballs of oxen in order to dissect this structure. In a properly preserved dissecting-room subject the eyeball can be admirably dissected after removal and distension with injected water. In view of the importance of arteriography in X-ray diagnosis, the labelling of vessels in such plates as Fig. 194 would be helpful.

The text, illustrations and production attain the high standards of clarity which we have come to expect in Cunningham. The book is warmly commended to students where anatomy dissection extends over 5 terms and would be most helpful to those preparing for the higher examinations.

P.G.L.

ALLERGY OF THE EYE

Ocular Allergy. By Frederick H. Theodore, M.D. and Abraham Schlossman, M.D., with chapters by William B. Sherman, M.D. and Roberts S. Coles, M.D. Pp. xviii+420. 111 figures. 96s. London: Baillière, Tindall and Cox Ltd. 1958.

More and more ocular affections are today being attributed to allergies. In some, such as an atopic conjunctivitis, the relationship is direct; in others, such as in phlyctenular disease, it is not so easily recognized; whereas in others again, e.g. uveitis, only an 'allergenic focus' can be invoked to explain the condition. All these aspects are fully discussed by the authors of this book and the clinical manifestations of ocular allergy described in a very easily readable text. The pitfalls in diagnosis and the conditions that can be confused are pointed out and the 13 tables are particularly helpful in summarizing the contents. In treatment the steroids have been extremely useful, but a warning is given that their indiscriminate use may be disastrous. Antihistamines have not generally been of much value and antihistamine ointments have produced so much sensitivity that the A.M.A. has disapproved their use. Every drug has at some time produced an allergic response, usually mild but occasionally fatal. The list of offending cosmetics is also a lengthy one. The subjects of vernal catarrh and conjunctival allergies are particularly detailed. This book can be recommended for study by every allergist and ophthalmologist. It is beautifully illustrated and the authors are to be congratulated in its production.

L.S.

THE ACUTE ABDOMEN

The Acute Abdomen. 2nd edition. By William Requarth, M.D. Pp. 313. 89 figures. 86.50. Chicago: Year Book Publishers, Inc. 1958.

This little monograph is a member of a series written for interns, surgical residents and practising doctors. Aetiology and treatment are considered, besides diagnosis. The approach to the subject is a novel one: an attempt to group conditions requiring immediate, delayed or non-operative treatment separately. There are also chapters on traumatic wounds of the abdomen, acute abdominal lesions in infants and differential diagnosis of acute gastro-oesophageal haemorrhage. Lists, preponderantly American, of references appear at the end of each chapter.

There is a profusion of roentgenograms, most of which are too small and could well be deleted to leave room for more detailed discussion of, say, appendicitis, electrolyte balance, or the restoration of body fluids. The discussion of some of the rarer conditions, e.g. volvulus of the caecum or spastic ileus, might also be deleted from this small monograph.

The chapter on intestinal obstruction is well written; almost nothing of importance seems to have been overlooked.

In acute appendicitis Rosving's sign is mentioned as a valuable help in diagnosis, and also Legat's sign for local hyperaesthesia and the so-called 'stage of illusion' in *perforative* appendicitis, when hyperaesthesia and pain suddenly disappear but the pulse rate rises.

The reviewer does not like the 'valuable diagnostic procedure' of peritoneal aspiration in 'all obscure cases of abdominal pain', by means of a 22-gauge spinal needle. In such cases it would seem safer and more practical to operate and see, than to aspirate and see.

The book is well printed and strongly bound. It reads easily and should be helpful to senior medical students interested in surgery.

G.C.A. v.d. W.

NON-TOXIC HYPERTENSION IN PREGNANCY

A Symposium on Non-Toxaemic Hypertension in Pregnancy. Edited by Norman F. Morris, M.D., M.B., B.S., M.R.C.O.G. and J. C. McClure Browne, B.Sc., M.B., B.S., F.R.C.S. (Edin.), F.R.C.O.G. Pp. xii+243. 78 illustrations. 35s. net. London: J. & A. Churchill Ltd. 1958.

For many years non-toxaemic hypertension in pregnancy has been a controversial subject to obstetricians and there has been a wide divergence of opinion between obstetricians and physicians. This book gives a detailed account of a symposium held on this subject at Hammersmith Hospital, where many eminent obstetricians and physicians gave their views. The book is well written and to the obstetrician who wishes to have a comprehensive account of most modern views on hypertension in pregnancy it is highly recommended.

It is stressed that there can be a marked difference in the systolic and diastolic blood-pressure readings according to the circumference of the arm. This is not often realized by the average obstetrician. Another factor brought out is the greater use of induction of labour, and it is emphatically stated that the last 2 weeks of pregnancy can be amputated without any deterioration in the condition of the baby. Although many obstetricians have lost faith in the hypotensive drugs, it is shown here that they can be of great value if used correctly.

Nevertheless, the obstetrician who is looking for a solution to all his problems on hypertension in pregnancy will be disappointed in this book. No definite plan for treatment or system of classification has been laid down. The question whether hypertension is a blood pressure over 120/70 or over 140/90 is not yet answered. No attempt has been made to decide on the definite point where you read the diastolic pressure, or whether the labile hypertension is of any importance. This, however, is one of the disadvantages of symposiums of such a nature, as no eminent authority wishes to compromise or set aside his views.

R.W.A.N.

BOOKS RECEIVED : BOEKE ONTVANG

The Medical Annual. A year book of treatment and practitioners' index. Editors: Sir Henry Tidy, K.B.E., M.A., M.D. (Oxon.), F.R.C.P. and R. Milnes Walker, M.S. (Lond.), F.R.C.S. Pp. xl+580+23+4. XLII Plates. 30 Figures. 42s.+1s. 9d. postage. Bristol: John Wright & Sons Ltd. 1958.

The Year Book of Radiology 1958-59. *Radiologic Diagnosis.* Edited by John Floyd Holt, M.D. and Fred Jenner Hodges, M.D. *Radiation Therapy.* Edited by Harold W. Jacox, M.D. and Morton M. Kligerman, M.D. Pp. 448. 336 figures. $10.00. Chicago: Year Book Publishers, Inc. 1958.

Clinical Obstetrics and Gynecology. Volume 1, no. 4. December 1958. *Symposium on Operative Obstetrics.* Edited by J. Robert Willson, M.D. *Symposium on Genital Cancer.* Edited by Daniel G. Morton, M.D. Published 4 times a year, over 1,100 pages. Illustrated. Sold by subscription only, $18.00 per year. New York: P. B. Hoeber, Inc. 1958.

Cardiac Arrest and Resuscitation. By Hugh E. Stephenson, Jr., M.D. Pp. 378. 31 figures. South African price £5 2s. 0d. St. Louis: The C. V. Mosby Company. 1958.

Breast Cancer. The Second Biennial Louisiana Cancer Conference. New Orleans, January 22-23, 1958. Edited by Albert Segaloff, M.D. Pp. 257. 43 figures. South African price. £2 2s. 6d. St. Louis: The C. V. Mosby Company. 1958.

Modern Treatment Yearbook 1959. Silver Jubilee edition. Edited by Sir Cecil Wakeley, Bt., K.B.E., C.B., LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S., F.R.A.C.S. Pp. xix+312. 2 figures. XXIII plates. 30s. London: Published for the Medical Press by Baillière, Tindall and Cox Ltd. 1959.

A Manual of Anesthetic Techniques. 2nd edition. By William J. Pryor, M.B., Ch.B. (N.Z.), F.F.A.R.C.S. (Eng.), D.A. (Eng.), F.F.A.R.A.C.S. Pp. 228. 75 illustrations. 27s. 6d.+1s. 1d. postage. Bristol: John Wright & Sons Ltd. 1959.

A Handbook of Medical Hypnosis. 2nd edition. By Gordon Ambrose, L.M.S.S.A. and George Newbold, M.B., B.S., M.R.C.S., L.M.M.S.A., D.R.C.O.G., D.C.H. Pp. xiii+276. 27s. 6d. London: Baillière, Tindall and Cox Ltd. 1958.

The Year Book of Urology 1958-59. Edited by William W. Scott, M.D., Ph.D. Pp. 364. 83 figures. $7.50. Chicago: The Year Book Publishers, Inc. 1959.

The Year Book of Ophthalmology 1958-59. Edited by Derrick Vail, B.A., M.D., D.Oph. (Oxon.), F.A.C.S., F.R.C.S. (Hon.). Pp. 407. 78 figures. $7.50. Chicago: Year Book Publishers, Inc. 1959.

International Text-book of Allergy. Edited by J. M. Jamar, M.D. Pp. 639. 17 figures. 110s. Oxford: Blackwell Scientific Publications. 1959.

The Hand: Its Anatomy and Disease. By John J. Byrne, M.D. Pp. xiv+384. 166 figures. 80s. Oxford: Blackwell Scientific Publications. 1959.

Abnormal Haemoglobins. A symposium organized by the Council for International Organizations of Medical Sciences, established under the joint auspices of UNESCO and WHO. Edited by J. H. P. Jonxis and J. F. Delafresnaye, CIOMS. Pp. ix+427. Illustrations. 45s. Oxford: Blackwell Scientific Publications. 1959.

Vascular Spiders and related Lesions of the Skin. By William Bennett Bean, M.D. Pp. xix+372. 130 figures. 63s. net. Oxford: Blackwell Scientific Publications. 1958.

Doctor Squibb—the life and times of a rugged idealist. By Lawrence G. Blochman. Pp. xii+371. $5.00. New York: Simon and Schuster. 1958.

CORRESPONDENCE : BRIEWERUBRIEK

DIE KUX-OPERASIE

Aan die Redakteur: Die anderdag het 'n oubaas met 'n uitgesproke parkinsonisme by my spreekkamer gekom. 'n Paar weke tevore is hy in die Transvaal agter die regterblad ,ge-Kux'.

Die pasiënt was niks beter nie en ekself niks meer ingelig nie, aangesien 'n beleefde brief aan die dokter wat die operasie gedoen het oor die aanduidings vir die operasie in die betrokke geval, geen antwoord tot gevolg gehad het nie.

Die enigste persoon wat dus by die hele prosedure gebaat het, was die Kux-chirurg, naamlik met 'n bedrag van £40.

E. J. Marais

Rouxstraat 18b
Bethlehem
1 Junie 1959

OCCUPATIONAL HEALTH IN INDUSTRY

To the Editor: In the *Journal* of 16 May, you published an article by Dr. Boris Serebro entitled *Occupational Health in South African Industry.*[1]

In the main I agree with what Dr. Serebro has to say about sick benefit funds, and the function of the medical practitioner within this framework. I am, however, in complete disagreement with Dr. Serebro's conception of the role of the medical practitioner in industry. I should like to draw your attention to what Dr. Serebro has to say in this connection:

'Today the doctor in industry must solve problems of a complex nature concerned with inter-human behaviour, among which priority is given to interrelations between management and labour

which, if maintained in an industrial organization facilitate communication and cooperation, ...'

'With discrimination he can sort out the various facts (in relation to worker's likes and dislikes re management), and can give definite indications of defects in managerial structure.'

I could also quote further extracts but feel these are sufficient to focus attention on the role of the medical practitioner in industry. Let us accept that the environmental conditions in industry are the concern of the doctor, then I submit that this refers to environmental conditions to a limited degree only. As I see it, the doctor is concerned with the physical demands a particular job makes on the individuals performing the job. Furthermore, he can advise on factors within the environment which may give rise to occupational disease, and on the preventive measures to be adopted. He is also concerned with the personal health of employees.

Once a medical practitioner devotes his attention to factors outside this field his contribution to management will be severely limited. Management in itself is a specialized field with an orientation which differs considerably from that of medicine. Management structures are not arrived at by trial and error, but are the result of the concentrated effort of various management specialists.

Considering for instance the field of human relations, I would remind Dr. Serebro that in management there are specialists who have received specialist training in this field, and whose opinions and findings are infinitely more reliable than those of a person not trained in this field.

The interpretation of the feelings and attitudes of employees can only be effected if the situation in which the employee works is understood. This can only be brought about by the concentrated study of management techniques, to the same extent that the medical practitioner studies the human body, in conjunction with the skill in observing behaviour in work situations and a thorough knowledge of motivation. I submit, therefore, that it is completely outside the province of the medical practitioner in industry to advise on, and diagnose, the ills of an organizational structure.

Whatever inherent weaknesses there are in labour-management, relationships are best left to be corrected by competent managers, who understand the situations in which these weaknesses arise. In my experience of dealing with problems of labour relations, I have not as yet found it necessary to have a doctor as arbiter. In fact problems of this nature would be as strange to the doctor as many medical phenomena are to the layman.

Without any doubt the medical practitioner could supply management with much information which would develop a *healthy* labour force, and it is in this sphere only that medical science can make its contribution to management.

F. P. Jacobsz, M.A., D.Phil.

9 Orwell Drive
Three Rivers, Vereeniging
5 June 1959

1. Serebro, B. (1959): S. Afr. Med. J., 33, 423.

SOUTH AFRICAN SOCIETY FOR CLINICAL HYPNOSIS

To the Editor: Allow me, through the medium of the *Journal*, to reply to correspondents who have written requesting more details about the International Society for Clinical and Experimental Hypnosis, and the newly formed South African division of this Society.[1]

In 1955, the Society for Clinical and Experimental Hypnosis (USA) decided to extend its activities by sponsoring a World Congress on Hypnosis and to draw to this Congress investigators from all over the world. This idea culminated in the formation of the International Society for Clinical and Experimental Hypnosis (ISCEH) in 1958 which now has divisions in all the continents and in 21 countries. Representatives of these countries—among whom are numbered many internationally distinguished contributors—serve on the Board of Directors which conducts the business of the Society. Among the key figures serving in the ISCEH are: Chairman, Dr. Bernard B. Roginsky (President, Academy of Psychosomatic Medicine, and Associate Physician, Montreal Jewish General Hospital); Secretary, Dr. John C. Watkins (Chief Clinical Psychologist, V.A. Hospital, and Lecturer, University of Portland, Oregon); and the Editor of the *Journal of Clinical and Experimental Hypnosis*, Dr. Milton V. Kline, of Long Island University and Seton Hall College of Medicine, USA.

The Board of Directors conducts its business largely by mail. Certain publications are controlled by the Board, but the international journal functions as an autonomous unit of the ISCEH. The ISCEH holds at least one scientific and general meeting every year.

Membership of ISCEH is open to members of the medical, dental and psychological professions. The Articles of the Society include the following Diplomas: Associate Membership, Full Membership, Fellowship, and Honorary Membership. These Diplomas are conferred after the Credentials Committee of the ISCEH has been satisfied of the necessary qualifications. Essential qualifications for Associate Membership are that the applicants should be members of the approved society or association for their respective professions, such as the Medical Association of South Africa; they should have had initial training in the use of hypnosis as demonstrated by the completion of an acceptable course of training or should present such other combination of training and experience as is deemed equivalent by the Credentials Committee of the ISCEH and they should be actually using hypnosis in general practice. For Full Membership applicants must have met requirements for Associate Membership and, in addition, present evidence of either scientific contribution or clinical experience proving abilities in the field of hypnosis.

Elevation to Fellowship is made by the approval and designation of the Board of Directors. This is a recognized honour bestowed by ISCEH. Honorary Membership may be granted by the Board to an individual who, although not fulfilling the requirements of the defined categories of membership, has distinguished himself in serving the cause of scientific research either directly or through service to ISCEH.

More information will, at a future date, be available to members who wish to apply for one or other of the Diplomas.

Various subdivisions sponsored by, and affiliated to, ISCEH include the Committee for Ethical Practices; Institute for Research in Hypnosis; Awards Committee—to designate annually the most meritorious research project or publication in hypnosis; and the American Board of Examiners in Clinical Hypnosis. At present the American Board of Examiners is incorporated under the Educational Charter of the Institute for Research in Hypnosis and will be governed by the University of the State of New York (Chairman, Dr. R. M. Dorcus, Professor of Psychology, School of Medicine, University of California). The American Board examines the competence of applicants with reference to their training and/or experience, thus setting a standard for examination for Membership to the ISCEH.

The South African Society for Clinical Hypnosis will form a national division of ISCEH. Its initial programme will be of a strictly academic nature, viz. organization of lectures, symposia, demonstrations and publications in hypnosis and allied sciences, so that members can attain the qualifications necessary to apply for enrolment in appropriate academic groupings of ISCEH.

A Northern branch of the South African Society[2] has been formed under the chairmanship of Dr. S. Levinsohn of Tara Hospital, Johannesburg, and a Southern branch under the chairmanship of Dr. M. Herman in Cape Town. Dr. Herman is at present overseas and will spend much of his time meeting key figures of the International Society to study methods of training, the role which universities and medical schools play in such cases, and forms of examinations.

Membership of the South African Society is open to all three fraternities but cannot, at this stage, provide any academic status. Names of colleagues who have applied for enrolment as diplomates of the International Society will be forwarded to the Credentials Committee of the ISCEH. Once the Constitution of the South African Society has been finalized, colleagues will be able to enrol formally, and partake of the academic programme and receive copies of the *Journal of Clinical and Experimental Hypnosis*.

M. Silbert
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1. Correspondence (1959): S. Afr. Med. J., 33, 264.
2. Passing Events (1959): *Ibid.*, 33, 525.